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THE PRACTITIONER

PRINTED BY EYRE AND SPOTTISWOODE,
HIS MAJESTY'S PRINTERS,
EAST HARDING STREET, E.C.

THE

PRACTITIONER

A Medical Journal.

EDITED BY

W. CECIL BOSANQUET, M.D.

JULY–DECEMBER, 1903.

"THE PRACTITIONER," LIMITED:
30, *HOLBORN, LONDON, E.C.*
1903.
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CATALOGUED
MAY 17 1904
E. H. B.

THE PRACTITIONER.

JULY, 1903.

1987

INTRODUCTORY ESSAY.

By T. CLIFFORD ALLBUTT, M.A., M.D., LL.D., D.Sc., F.R.C.P., Hon. F.R.C.P.I., F.R.S.,

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THE Editor of THE PRACTITIONER has done well to devote successive numbers of his magazine to the discussion of a particular disease; to hold, as it were, a public disputation upon each and several of them. On this occasion, the discussion of Gout, he has done me the honour to invite me to read the collected theses beforehand, and to take upon me the function of a chorus. He would ask a plain man to say what he makes of this discussion; if the collected essays show the subject more in the round than before? if from them a spectral outline has gained some solidity, or vague apprehensions some definition? From Sydenham's day to our own—to penetrate no farther into the past—we have not failed to talk about gout; how much more do we know of it than he did? When that able physician who knows so well to combine abstract science with practice—Dr. Luff, when Dr. Luff tells us that "the gouty individual is one whose general metabolism is instable, and that this instability may be present in one or more of the great physiological systems—the digestive, the nervous, the circulatory, &c."; and when Dr. Bannatyne, out of a rich experience at Bath, tells us that "in gout we have a disease which may give rise to almost any symptom, or affect almost any organ or function," we are pleased to think how thankful Sydenham would have been for a vision of these explicit results of two hundred more years of progress! It is something at any rate to know precisely what you are going to talk about. This merit, however, belongs more conspicuously to Dr. Haig than to

Dr. Luff or to Dr. Bannatyne. The plain man dislikes evasions, qualifications, and subtleties; and the dogmatism of Dr. Haig, which has nothing sophistically subtle about it, no less than his sincerity and earnestness, stir him and captivate him; until he finds that the prophet—a name I will now leave in print as it has escaped from my pen—is exhorting us to fare by a very strait gate indeed. Cast off your dietetic sins, he says; "and when the change has been made and become general, future generations will look back on our present records of sorrow, suffering, and degradation with amazement." "Abstain from flesh and tea," and good shall be your portion; indulge in them, and you shall fall victims—not only to gout and rheumatism, but also to chlorosis, epilepsy, hysteria, Bright's disease, mental disease, Raynaud's disease, pernicious anæmia, diabetes, and obesity. The plain man is not unwilling to go so far as to denounce "high teas"; but that tea and all flesh, creatures to which he thinks he has owed much sustenance, and which, at the worst, are very comfortable poisons, are to be utterly abjured, is a discipline for which he does not yet feel fully ripe. Dr. Luff reassures us by supposing that Dr. Haig and his congeners have poor insides, and that the diet they allow themselves is as good as, physically speaking, they deserve. However this may be, the plain man certainly likes dogma, and he likes it with no gnostic refinements about it; so Dr. Haig will not lack disciples.

When we leave Dr. Haig we feel that the rest of the company, unless it be Dr. Toogood, are trimmers; and Dr. Toogood in his shrewd and pleasantly written article does not make so ruthless a demand upon us as Dr. Haig does. Dr. Haig is definite by amplitude of inclusion, Dr. Toogood by a narrower precision. Under Dr. Toogood's eyes in Lewisham Infirmary a large pathogenetical experiment is tried, under conditions of more than ordinary simplicity. He sees the "mean white" under two adversities, poverty and gout; both of which he discovers to be "artefacts," and due to beer. Probably wine would do equally well; but the indigent cannot have everything. Spirits do not attain both ends; not a few of his patients try, especially women, but they achieve poverty only. Laundresses, however, who cling to beer carry off the double event. The aboriginal poor, who cannot get even beer, have to submit to a chillier penury, undiversified even by gouty fires. For Dr. Toogood gout is a disease, chiefly of the joints, consisting in encrustation of the parts affected by urate of soda; that his field of observation has its limits he well knows; but his results are the more telling, and perhaps the more true. At any rate he carries us far away from the conception of gout as a masquerade in which all bodily diseases, or almost all, may play their parts.

Now if gout is uratic arthritis, at any rate we know where we are; when we quit this rock and, trusting to sundry pilots, try to plant the flag on a motley archipelago of dwindling kidneys, torpid livers, plethoras, flatulent dyspepsias, megrims and melancholies, we are sailing on uncharted seas. A patient in gout surely ought to be more or less gouty; and if we perceive other disorders consistently hovering about his gout, premonitory, contemporary, or consequential, as the case may be, we may know them by their associates, and include them cautiously in the same predicament; but to say of many and many a patient that he is gouty when he never had a uratic arthritis in his life, much as he may have prayed for it, seems a topsy-turvy nosology. It may be urged that he has disorders identical with those which hover around the arthritic man; but the identity is surely an identity of negatives rather than positives, of common disorders rather than of specific characters or consistent groups of characters. Dr. Walker Hall is pursuing the only right way of unravelling the skein of nutrition and excretion, but his researches seem to have no concentration upon gout; unless we take gout to mean any perturbations of the rise and fall of the fountain of life. We cannot even assume that gout is some particular crookedness in the ascent or descent of living processes; for it is at least as likely that it may consist in a vulnerability of the fibrous structures, whereby they are obnoxious not only to uric, but also to rheumatic and other invasions. Dr. Haig is right in classing rheumatism and gout together, if we go no farther than to say that joints which in their youth are prone to rheumatism are prone in later years to gout. Dr. Toogood's note on uratic joints in marasmus also suggests that a susceptibility of these structures may be a cause of gout. Again, we are far from sure that gout is not due to some extrinsic element. The peculiar geography of gout scarcely receives sufficient attention; some such article, with its correlative descriptions of diet and habits, would have been welcome in the present collection. Dr. Crombie indeed does something to fill this gap. Furthermore, the effect of microbic infection on the precipitation and concretion of uric acid in the pelvis of the kidney has not received the attention which, in this connection, it deserves. And yet again, as in diabetes, I suspect that a copula or "amboceptor" between the proteid molecule and the passing carbohydrate is in defect, so, perhaps, in gout some internal secretion may fall short of such a factor. Till we know, then, whether gout is a mere shortcoming in ordinary metabolism, or a peculiar perversion of it, or a static susceptibility of fibrous tissues, or a defect of some ferment or copula, or, as Sir Dyce Duckworth urges, a perturbation of some nervous centre, or again some factor from without, it appears to me we must confine this name to the uratic precipitations which we know, and which can serve us as a touchstone; and as to all other vague "acidities," flatulencies, megrims, and biliousnesses of whatever occult kind, be content to treat them empirically, awaiting the results of the analysis of our nutritive life upon which bio-chemistry is actively and hopefully engaged. Meanwhile Dr. Walker Hall supports "without doubt" what was substantially Murchison's teaching concerning the part of the liver in destructive functions.

All the contributors seem agreed that an eczema may be gouty, though they shrink from Bazin's minute differential diagnosis of the kind. If there be an eczema of gouty causation, it is probable that gout is but the initiative factor—by deposit of urate of soda in the skin? but that once established it progresses by its own inertia and by infection from without. For my part I think "gouty eczema" is in large part an eczema of dry skins, in which fire-heat, scratches, and the like readily produce lesions which lay the skin open to extrinsic infections.

In one important respect I lag behind most of the disputants. I do not find arterial pressures to rule much higher in gouty persons—*i.e.*, in the subjects of uratic arthritis—than in other persons of middle and later life. I am glad to number still among my living friends not a few who have been subject to what the plain man calls gout for many a decade, yet who never had arterial hyperpiesis, and are still hale if not always active. Happily, diverse as our counsellors' doctrines may be, they nearly all agree upon the main lines of practice; whether dietetic, pharmaceutical, or balneological. I say "nearly all," for their common-sense eclecticism, reinforced, as it is, by the weighty judgment of Sir Dyce Duckworth, must seem to the ardent spirit of Dr. Haig a mere time-serving. He will point to Dr. Toogood's remark that too early an abandonment of his convalescents to flesh and fish means a relapse. Those who are of the stuff of martyrs will follow Dr. Haig; but I fear the plain man will tarry in the *via media*.

THE OCCURRENCE OF GOUT IN THE UNITED STATES.*

By THOMAS B. FUTCHER, M.B. (Tor.),

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THE general impression has prevailed both within and outside the United States that gout is a rare disease in this country. I have failed to find any published statistics in America enabling one to draw a comparison between the frequency of the disease in this country and in England or the Continental countries. A careful search of the *Index Medicus* and *Index Catalogue* has failed to give assistance in this direction. Clinicians in this country have usually taught that gout is a very rare affection. Jacob Bigelow,[1] of Boston, in the second edition of a collection of essays entitled *Nature in Disease*, published in 1859, devoted a chapter to "Gout and its Treatment," and here as well as elsewhere in the volume makes statements which lead one to infer that cases of gout were not extremely infrequent in his practice. The English authorities, practically without exception, have stated that gout is of rare occurrence in the United States. Thus, Sir Dyce Duckworth,[2] writing in 1890, makes the statement that "in the United States of America gout is practically unknown. A few cases are met with in the large cities, but, according to Dr. Da Costa, of Philadelphia, the disease has not yet developed among the increasing population." Ewart[3] speaks of the United States and Canada as possessing a considerable immunity from declared gout.

It has been thought that the habits and environment of the people of the United States have been of such a nature as not to be conducive to the development of gout. In England, where the disease is generally conceded to be more prevalent than in any other country in the world, the social conditions and habits have been those regarded as being most favourable for the production of gout. There has for many generations been a large leisured class who have been over-indulgent in eating and drinking, and who have not taken sufficient exercise. Sir Dyce Duckworth believes that the supposed infrequency of gout in the United States is due to the following conditions:—"The ancestry, diet, and habits of the citizens of the United States for the most part are such as will long prevent the onset of gout in that country. The immigration of Irish, Scottish, Scandinavian, and German people into that country peoples it mainly with a stock void of gouty heritage. The good climate and the active open-air life of the people will tend to avert goutiness. But little wine is used, the beer is light, and much water and weak tea is taken. The alcohol chiefly used or abused is rye whisky." These conditions undoubtedly prevail, and are in all likelihood factors in preventing gout from being more prevalent than it is. There are in this country, as in every country, those who attribute to a so-called uric-acid diathesis every vague pain or headache. There are various springs that are Meccas for such patients. The uric-acid diathesis here, as elsewhere, is a scrap-basket for all improperly diagnosed cases. Such cases are not included in the series here reported.

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* From Prof. Osler's Clinic at the Johns Hopkins Hospital, Baltimore.

Notwithstanding these generally expressed views as to the comparative infrequency of gout in the United States, there is no question but that the disease is much more prevalent than physicians suppose. In considering the comparative infrequency of gout in this country the failure to recognise the affection must be taken into consideration. Our experience with cases admitted to the Johns Hopkins Hospital has amply shown that many are mistaken for rheumatism even when well-recognisable tophi are present about the joints or in the ears. The disease is not an unimportant factor from an insurance point of view. The *Report of the Mortality Records of the Mutual Life Insurance Company of New York* for 56 years (from 1843 to 1898) shows that there were 50 deaths from gout out of 46,525 mortalities. The best method of determining the relative frequency of the disease in the United States as compared with southern England, where cases are more numerous than in any other part of the world, is to compare the number of cases admitted to a general hospital for acute diseases in a large American city with the number of cases admitted to a similar institution in London. Thus, for the first 14 years since the Johns

Hopkins Hospital was opened, from May 15, 1889, to May 15, 1903, there were 41 cases of gout admitted to Dr. Osler's wards out of a total of 15,697 medical admissions, or, in other words, 0·26 per cent. of the cases. For a similar period of 14 years, namely from 1888 to 1901 inclusive, there were 124 cases of gout admitted to the medical wards of St. Bartholomew's Hospital,[4] London, out of a total of 33,356 admissions, or 0·37 per cent. of the medical admissions. That is, in London, the home of gout, the cases are a little less than one-third more frequent than they are in Baltimore. There is particular reason to believe that gout is more frequent in Baltimore than in any other large American city. This disparity is not at all marked, and it is ample evidence that gout is not a rarity in the United States.

In addition to these 41 cases of gout admitted to the medical wards, there was one of chronic gout with suppurating tophi admitted to Prof. Halsted's wards for surgical treatment, making altogether 42 cases admitted to the hospital wards. Hardly a month passes without one or more cases of gout coming under observation in the Out-patient Department also. The 42 cases analysed in this series will serve to illustrate the main features of gout as they are manifested in this country. We will consider first what influence the usual ætiological factors of heredity, alcohol, food, and lead have had in the causation of the disease in these cases.

PREDISPOSING ÆTIOLOGICAL FACTORS.

Hereditary Tendencies.—In only four cases was there a definite statement that a parent or grandparent had suffered from gout, or in only 9·5 per cent. of the cases. This is in striking contrast with the statement that statistics show that in from 50 to 60 of all cases the disease existed in one or more ancestors. In 10 other cases there was a history of arthritic attacks described as "rheumatism" occurring in the immediate antecedents. The probability is that the majority of these cases were gout rather than rheumatism, as patients usually have an aversion to the statement that there is gout in the family. Putting the most liberal interpretation on these cases described as rheumatism and considering them as cases of true gout, it would give us 14 cases with a amily history of gout, or 33·3 per cent. of the cases. This evidence would seem to go to show that gout in this country is in the majority of cases freehold, not copyhold, to use the division of old Sir William Browne.

Alcohol.—Alcohol has always been considered a potent ætiological factor, and the form is probably as important as the quantity. Fermented liquors, such as wine, beer, ale and porter, are much more injurious than the distilled liquors, as whisky, rum, &c. Forty of the 42 patients used alcohol in some form or other. Regarding the other two there is no record as to their alcoholic habits. Two of the 40 patients used whisky alone. We have made it a point to ask carefully in all gouty cases regarding their alcoholic habits, and there is a preponderance in the number who have used fermented over those who have used distilled liquors in excess. Beer has been the beverage mainly used, although nearly every beer-drinker has drunk whisky also, either moderately or in excess; none of the patients used wine. Alcohol appears to have played an important part in the ætiology of these cases, although the lighter beers of this country are not so likely to predispose to the development of the disease as are the heavier beers and ales of England.

Food.—The character and quantity of the food can hardly be said to have been a powerful causative agent. The patients did not belong to a social class capable of obtaining abundance of highly-seasoned, nitrogenous food. Nor were they able to lead sedentary lives, as the majority belonged to the labouring classes and required to work for a livelihood. Eleven stated that they were heavy eaters, six being particularly meat-eaters.

Lead.—How and why lead proves to be such an important factor in the production of gout is still a question requiring explanation. Although Musgrave, Huxham, and Falconer (1772) had called attention to this association, it remained for Garrod[5] to emphasise this point. He found that at least one in four of the gouty patients who had come under his care in the hospital had at some period of their lives been affected with lead, and for the most part had been either plumbers or painters. Garrod showed that the blood of patients suffering from lead-poisoning contains an excess of uric acid, and it is generally held that the increased liability of lead-workers to gout is due to the metal producing arterio-sclerosis and chronic nephritis with consequent renal inadequacy.

In only one of our cases was there definite evidence of the patient suffering from lead-poisoning. The patient was a painter, and was admitted to the hospital suffering from lead-colic. There were, however, in all eight cases whose occupations exposed them to lead-poisoning. Five of these were painters, and three were tinners. Thus in 19·4 per cent. of the cases lead was undoubtedly an important ætiological factor, a percentage very close to that noted by Garrod. It must then be concluded that lead is also an important causative agent in the United States, although not apparently quite so potent as in England.

Nationality.—In considering the frequency of gout in this country it is important to note the nationality of the victims. Thirty-two of the 42 patients were native-born Americans; 5 were born in Germany; 3 in Ireland, and 1 each in Canada and Finland. It will thus be seen that only a small number of the cases occurred in immigrants. In none of these 10 foreign-born patients was there a definite history of gout in the ancestors, although in 4 there was a history of "rheumatism."

Age and Sex.—The ages of the patients on first admission to the hospital, according to decades, was as follows :—From 1 to 10 years, no cases; 11 to 20, 1 case; 21 to 30, 1 case; 31 to 40, 11 cases; 41 to 50, 13 cases; 51 to 60, 10 cases; 61 to 70, 5 cases; 71 to 80, 1 case. The greatest number of cases (13) occurred between 41 and 50 years, or in the fifth decade. The youngest case was in a boy 17 years old. This is an extremely early age for gout to manifest itself. Gairdner has reported fits of gout in infants at the breast. Cases are extremely rare under 20 years. The rarity of the disease in children and minors is illustrated by Scudamore's statistics of 515 cases. Only 4 occurred before the age of 17, the youngest being 8 years old. There were but 13 in the first two decades. When gout appears in very young individuals it nearly always is inherited. The oldest patient in our series was 75 years of age. His first attack, however, had occurred at the age of 48.

Gout is much more common in males than in females. Women appear to possess a relative immunity. Of the 124 cases at St. Bartholomew's Hospital in 14 years there were 100 in men and 24 in women. It would appear that females in the United States are even less frequently affected. Our 42 cases were all in males.

Race.—The negro appears to possess a relative immunity. All our cases, with one exception, were in whites. This would give the proportion of negroes to whites affected as 1 to 41, whereas the ratio of coloured to white admissions is about 1 to 7.

Social Position.—The cases nearly all occurred in individuals in the lower walks of life. Only 3 of the 42 were treated in the private wards, the remainder having been admitted to the public wards of the hospital.

Occupation.—The occupations of the patients were varied. As having a special bearing on the ætiology of the disease, it may be recalled that 5 were painters and 3 tinners. Three were bar-tenders or saloon-keepers. The frequency with which the latter are liable to be heavy drinkers renders them also specially prone to gout.

CLINICAL TYPES.

Acute Gout.—Three cases were admitted during the initial arthritic attack. These were the only cases of acute gout in the series.

Chronic Gout.—The remaining 39 cases were all chronic, the patients having had several attacks before coming under observation. The shortest duration of any of the cases in this group being one and one-half years; the majority, however, having lasted for a much longer period. These patients were all admitted suffering from acute acerbations.

Tophaceous Gout.—In 19 of the 39 chronic cases, or 48·7 per cent., tophi were present. The existence of the tophi made the diagnosis certain, and indicated the chronicity of the disease in these cases. The distribution of the tophi was as follows:—In 17 cases they were present over the cartilage of one or both ears. They were present in four cases in the subcutaneous tissue over the elbow-joints, and in one over the patella. In these situations they closely resembled the subcutaneous fibroid nodules of rheumatism. In 4 cases they occurred about the finger-joints, and once over the big-toe joint. One patient had a huge tophus measuring about

5 by 3 cm. over the left tendo Achillis, and another had a large group of them over the sacrum. In 4 cases a bursitis occurred, resulting from the deposition of sodium biurate in and about the bursal sac. In 2 cases the bursa over the olecranon was involved, and in the 2 other cases the prepatellar bursa was the seat of the suppurative inflammation. In 3 cases the characteristic acicular crystals of sodium biurate were obtained from the contents of the bursæ. In 2 cases there was suppuration of the tophi about the finger-joints, and once of a tophus over a big-toe joint. Altogether suppuration of the tophi occurred in 5 cases.

Cardio-vascular system.—The peripheral vessels, particularly the radials, showed a more or less marked degree of sclerosis in 29 cases. The heart was moderately enlarged in 5 patients and there was a mitral systolic murmur, indicating a certain degree of insufficiency, in 8 cases. In only 2 cases was there very definite evidence of marked myocardial disease. This patient had a persistent embryocardia. One of the fatal cases died with a terminal serofibrinous pericarditis.

Urinary System.—It is interesting to note the great frequency with which there were evidences of changes in the kidneys in the series. In 15 of the cases the specific gravity of the urine was persistently below 1015. Albumen was present, usually in small traces in 32 cases, and hyaline or finely granular casts, or both together, in 26 cases. We may say, then, that in 32 of the 42 cases, or in 76 per cent. of the patients, there were strong evidences of more or less serious organic renal disease. Considering the persistently low specific gravity of the urine in 15 of the cases, the probabilities are that the disease had advanced to the chronic interstitial stage in more than 50 per cent. of the cases in which albumen was present.

In the two fatal cases of the series the kidneys were found at autopsy to be very much contracted and the seat of advanced chronic interstitial nephritis. One of the cases died of uræmic coma.

I would like to help to correct the impression, apparently held by many practitioners, that a uric-acid sediment in the urine indicates a gouty tendency. The power of the urine to hold uric acid in solution is dependent on so many factors, such as the amount of pigment and the percentage of salts present, that but little significance can be attached to the existence of a uric-acid sediment. In none of the 42 cases of gout was a uric-acid deposit made out on either gross or microscopic examination even in the acute attack. This affords abundant evidence that a uric-acid sediment in the urine is not by any means a necessary accompaniment of gout. Glycosuria was not present in any of the cases.

COMPLICATIONS.

Bursitis occurred in four cases. The prepatellar bursa was inflamed twice, and that over the olecranon twice. In one of the cases it was a suppurative bursitis, and in three instances the bursæ required to be opened. Unilateral parotitis occurred once. This is a very rare complication, but has occasionally been described. Serofibrinous pericarditis was a terminal event in one case. This patient illustrated some of the remarkable features of retrocedent or visceral gout. The acute joint-symptoms had practically subsided when marked abdominal symptoms manifested themselves. There was intense pain in the umbilical region with marked abdominal distension. These lasted for a week, gradually getting worse. His symptoms were believed to be due to possible intestinal obstruction or to peritonitis, and his condition seemed to demand operation. Nothing was found at operation to explain the enormous intestinal distension. The patient died a few hours later, the autopsy revealing a pericarditis, and small contracted gouty kidneys. There were but few skin-complications. There was one case of acute eczema and one of herpes zoster.

MORTALITY.

Two patients in the series died in the hospital. The main features at autopsy in each case have already been briefly referred to.

DIAGNOSIS.

I am quite sure that the apparent infrequency of gout in the United States is due to failure on the part of the profession to recognise the disease. The affection is usually mistaken for rheumatism. The diagnosis in the series of cases here reported was established by the presence of tophi, or by a careful consideration of the arthritic manifestations, together with the ætiological factors present in the history. In a certain percentage of the cases, with a history of recurring attacks of arthritis limited to the big-toe or tarsal joints, and occurring in a member of a gouty family, there is little difficulty in establishing a correct diagnosis, even in the initial attack. Unfortunately, however, as our experience with this series has shown, the diagnosis is not so simple. In many cases the arthritic attacks are not limited to the feet, and there may be a multiple arthritis involving several of the other medium-sized and larger joints, and it is these cases that are liable to be mistaken for and treated as rheumatism. This was the case in four of our series, the cases having been repeatedly admitted and discharged with a diagnosis of rheumatism. Eventually the cases returned with characteristic tophi on the ears or about the joints, revealing without a shadow of a doubt the true nature of the affection. Two of these cases had subcutaneous tophi in the region of the elbows when they were being treated as rheumatism, but these nodules were taken to confirm this diagnosis because they were believed to be rheumatic fibroid nodules. Sections of these, after their removal, subsequently showed them to be tophi, their centres containing deposits of sodium biurate. In a case of arthritis with doubtful diagnosis accompanied by persisting subcutaneous fibroid nodules, the removal and histological examination of one of the latter may absolutely settle the diagnosis. As our statistics have shown, tophi are by far most frequently found in the ears, and Dr. Osler has strongly emphasised the importance of the careful inspection of the latter in all cases of multiple arthritis of doubtful origin. The contents of any whitish, chalky-looking body occurring over the cartilage of the ear in such cases should be examined microscopically. The finding of the characteristic acicular needles of sodium biurate immediately settles the case as one of gout. Small fibroid nodules, Woolner's tip, and minute sebaceous cysts may be mistaken for tophi, but the microscope serves to differentiate them.

The character of the uric-acid curve can be used in institutions to assist in the diagnosis, but this is not possible in private practice. The occupation of the patients should be carefully inquired after. The occurrence of recurring arthritic attacks in painters, plumbers, brewers, and saloon-keepers should lead one to strongly suspect gout.

SUMMARY.

1. The apparent infrequency of gout in the United States is due in large part to failure to recognise the disease.

2. Out of 15,697 medical cases admitted to Dr. Osler's wards at the John Hopkins Hospital during a period of 14 years there were 41 cases of gout, of 0·26 per cent. of the total number of medical patients. For the same number of years at Saint Bartholomew's Hospital there were 124 cases out of a total of 33,356 medical admissions, or 0·37 per cent. of the medical cases.

3. The ratio of admissions of gout to the two hospitals is respectively a little greater than as 2 is to 3; or, in other words, the admissions of gout to a general hospital are a little less than one-third more frequent in London than in Baltimore.

4. All of the 42 cases were males. There was only one coloured patient in the series. The negro race appears to possess a relative immunity against the disease. The largest number of cases, 13, occurred in the fifth decade. Of especial interest is the fact that 32 of the patients were native-born Americans.

5. The majority of the cases appear to have earned rather than inherited their gout. Alcohol and lead seemed to be the most potent ætiological factors.

6. Thirty-nine of the 42 cases had reached the chronic stage before they came under observation. In 19 of the cases tophi were present.

7. Among the most interesting complications may be mentioned four cases of gouty bursitis; one case of parotitis, one of pericarditis, one of retrocedent gout with symptoms simulating intestinal obstruction.

8. There was evidence of disease of the kidneys in the majority of the cases. Albuminuria occurred in 32 and hyaline or granular casts in 26 instances.

9. Arterio-sclerosis of varying degrees was present in 29 cases and a mitral systolic murmur in 8.

10. The difficulty of differentiating the disease from rheumatism was illustrated by the fact that four of the cases were repeatedly diagnosed as the latter on their early admissions to the hospital, the appearance of tophi later revealing the true nature of the disease.

11. The series illustrates the great importance of examining the ears and the vicinity of the joints for the presence of tophi in all cases of multiple arthritis of doubtful origin.

BIBLIOGRAPHY.

[1] Jacob Bigelow: *Nature in Disease*, pp. 57 and 134.
[2] Sir Dyce Duckworth: *A Treatise on Gout*, 1890, p. 340.
[3] Ewart: *Gout and Goutiness*, 1896, p. 28.
[4] *St. Bartholomew's Hospital Reports*, Vols. for years 1889 to 1902.
[5] Garrod: *Gout and Rheumatic Gout*, third edition, 1876, p. 237.

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GOUT IN THE TROPICS.

By ALEXANDER CROMBIE, C.B., M.D.,
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MOST of the authorities on tropical diseases are silent with regard to gout. This arises from the fact that the manifestations of gout offer no peculiarities in hot climates, and therefore do not call for special notice in treatises devoted to diseases peculiar to the tropics, and also because they are less frequent. Writing in 1837, Dr. Corbyn pointed out that gout is scarcely known among the inhabitants of warm climates, and Dr. Irvine, in his *Medical Topography of Ajmeer*, says, "I have heard of gout occurring among some of the rajpoots who drink hard, but I never met with such cases." And Chevers (*Diseases of India*) says, "Although I have seen one or two very moderate cases of this disease among Europeans, and have heard of a few others, I think that, with all its sanitary faults, Calcutta is an earthly paradise for those cautiously-living people whom gout threatens but has not crippled," and he is of opinion that a young Englishman, born of gouty parents, but still free from joint-disease, may enter upon a career in Lower Bengal with a strong hope that, with prudence, he may escape the threatened evil.

I have seen, in private practice, a good many cases of distinct though mild gout, of a classical character, among Europeans, Jews, and well-to-do natives in Calcutta, where I found a very considerable demand for a well-known specific; and I once saw a missionary, who had spent his whole life in India, with well-marked chalk-stones; but there is, I think, no question that the disease is much less frequent in that part of India, and probably in other hot climates, than it is in Europe among the same class of patients. It is very rarely met with in hospital practice in that country.

On the other hand the irregular manifestations of the diathesis are by no means rare in the European who has become exhausted by long residence in a hot climate, if he inherits a predisposition to gout, and has indulged freely in the luxuries of the table. It then takes the form of gouty eczema of the legs and scalp, of acid and flatulent dyspepsia, gastric catarrh, and congestion of the liver; and in younger men with a strong hereditary taint, of headache, with high blood-tension, and inability to stand exposure to the sun.

In looking for an explanation of the comparative infrequency of the manifestations of gout in hot climates, I think it will be found in the lower tissue-metabolism which is required to raise the temperature of the body through a smaller number of degrees, and among natives, who adhere to the rules of their religion, in the less nitrogenous nature of their diet. In a temperate climate the body-temperature is maintained at some 40° F. above the atmospheric temperature, whereas in a hot climate it may be only 5, 10, or 15 degrees above that of the air. Less tissue-change is therefore necessary. Evidence of this lessened metabolism is found in the lower specific gravity of the urine, which ranges in Europeans from 1015 to 1018, and in natives from 1008 to 1014, while at the same time the quantity is diminished to some extent, averaging about 1250 cc. against 1500 in Europe. From these figures it may be calculated that the excretion of urea is lowered from 33 grammes in Europe to about 22 grammes in Europeans in a hot climate, and to about 17 grammes in natives; and we may assume that there is a corresponding facility in dealing with uric acid and other products of tissue-change, on the excess of which the proclivity to gout depends. On their return to reside permanently in England, Europeans who have enjoyed an immunity from the disease in India frequently suffer from gouty attacks under the changed conditions of life.

I do not think that the diminished liability to gout can be attributed in the case of Europeans living in a hot climate to any difference in their habits as regards diet, for there is no marked difference in this respect. The European living in Calcutta has three meat-meals a day, often of the richest quality; and, although he probably consumes less wine and alcohol generally than his brother in England, he is by no means abstemious. The native, on the other hand, has a dietary which is largely farinaceous, and consists chiefly of rice, pulse, fish, green vegetables, and milk. With him the tendency is not to gout but to diabetes, which is very common in well-to-do natives over forty years of age; so much so that I was accustomed to say that diabetes is the gout of the Hindus. It chiefly attacks, like gout in Europe, those whose occupation is sedentary and mental, and, like gout, it selects those especially who are engaged in the administration of the law.

GOUT AS IT OCCURS AMONGST THE POOR.

By F. S. TOOGOOD, M.D. (Lond.), D.P.H.,

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POVERTY being a relative term, it would be better for the gout-stricken subject if he were just the amount poorer necessary to prevent his acquisition of malt-liquor. I doubt if the really poor, meaning thereby those in the clutches of downright grinding poverty, ever develop gout. I have never met with a case in genuine frequenters of the casual wards, who, as a class, would be perhaps the poorest in the metropolis. The ordinary victim of gout, as we know him in the infirmaries, is poor because of his habits, and his disease drives him to seek relief at a State hospital, whereas with greater temperance and more thrift he might have either escaped the disease altogether or have endured his earlier attacks in his own home.

Sex.—The disease is much more common amongst men than amongst women. This I believe to be due mainly to the greater sobriety of women, but also to the circumstance that, when women become habitual drinkers, they usually affect spirituous liquors, because they more rapidly feel the elevating effect of the alcohol, and because they do not take large draughts of liquid to quench an "occupation" thirst.

Women do not seek admission during the early acute attacks of the disease, because their presence at home is absolutely essential for the purpose of keeping an eye upon the house and the children. The earlier admittance of the man is explained by the custom of the Poor Law authorities of granting out-door relief to families without much demur when the breadwinner is in hospital.

The case-ratio is about twenty males to one female: the admission-ratio is much higher, some men being admitted several times during a year! A woman rarely applies for admission until she is in the helpless chronic late stage of the disease.

It is difficult accurately to estimate the effect of *heredity*. In the great majority a history of joint-trouble in the father or grandfather or both is easily elicited; but unless the actual presence of chalk-stones was noted, it is usually impossible to separate a family history of gout from that of the various forms of rheumatism. Not infrequently, however, where the grandfather has been known to have had gout, the father has been free.

Age.—The majority of cases occur for the first time between 40 and 50 years, but some as early as 35; and when associated with lead-poisoning it may occur even earlier than this.

Occupation appears to influence the production of the disease to a very slight extent. No class of workman seems to be specially or particularly subject to attack; nor does the nature of the mechanical work performed, involving special strain upon certain joints, have any effect in determining the first joint to be attacked. The metatarso-phalangeal joint of the great toe is universally the first to be affected; and this is the case even when the hands are subjected to much more strain in performing trade labour than the feet are in walking. This is particularly shown in the poorer class of cobblers who work long hours, who use their hands continually, and whose walking consists merely of a few journeys up and down stairs daily, with the evening perambulation to the favourite public-house.

But increased use of the feet in *walking* unaccustomed distances certainly induces an attack. Many cases have come under my observation where men, who, having led a sedentary life all the year in London, had proceeded in the autumn to walk to the hop-fields, have had their country holiday rudely interrupted by an acute attack occurring before they were able to reach their picturesque and aromatic destination. One man I remember who made the attempt several years in succession and was invariably pulled up by his enemy. He was eventually successful, but only because some circumstance induced an attack during the summer, and so cleared his system sufficiently to enable him to accomplish his journey.

When, however, the disease commences to attack other joints, the influence of occupation is markedly shown. Cobblers are especially liable to have the little finger of the left hand affected, this being subjected to great strain in pulling the thread whilst sewing. The right little finger is not affected, because the thread is twisted round the handle of the awl, which is held in the right hand.

Coachmen are especially affected in the interphalangeal joints and wrists; the hands being attacked later, but much more severely, than the feet. The story of a coachman's downfall is the same in every instance. There is the struggle against the increasing stiffness of the hands; the endeavour to retain the situation by the payment of outsiders to perform all duties save the actual act of driving; then some small accident which reveals the crippled condition of the driver, and his consequent discharge. After this there is casual employment at a livery-stable or public-house yard, where the increased facility of getting beer induces frequent attacks with consequent sojourns in the Infirmary, until finally the distorted joints can no longer earn sufficient to keep body and soul together, and the victim becomes a permanent inhabitant of the infirm wards of a workhouse.

Laundresses form the bulk of the females who come under the care of the Poor Law authorities suffering from gout, and here, again, the determining cause is beer. Many laundries, especially of the lower class, supply their workers with a certain number of half-pints daily, and the nature of the work appears to induce a constantly-recurring thirst. Ironers develop a characteristic deformity of the fingers of the right hand, almost exactly resembling the ulnar deviation accruing in rheumatoid arthritis. This is caused by the lateral pressure of the iron upon the fingers. It does not occur in the left hand, and the history serves to distinguish between the two complaints.

Enough has been said to indicate clearly the fact that the *consumption of malt-liquor* is the chief, if not the only, cause of the production of gout; and although many spirit-drinkers are admitted from various causes to the Infirmary, they do not suffer from gout. I make it a rule to enjoin absolute abstention from all forms of alcohol upon gouty patients; but knowing how difficult it is for some persons to refrain entirely, I tell them that if they find themselves so placed that they must take something, then gin is the least harmful to them. Those who follow either course do not again develop an attack.

The beneficial effect of entire teetotalism, combined with plain wholesome food, is strikingly shown in those cases who, having been absolutely crippled and broken down by the disease, have become permanent inmates of an infirm ward. The distorted and stiffened limbs do not regain their shape or use, but the patients do not develop any fresh attacks so long as they remain with us, where the consumption of beer is effectively prevented. Should their friends, however, "take them out" for a few days the unwonted indulgence of "a few glasses" frequently induces an acute attack.

Wine as a staple article of drink is not known amongst the English poor. Some unusual occasion for rejoicing may be signalised by the consumption of a bottle of sweet mawkish port, but clarets, white and sparkling wines are practically unknown.

Food is not an important factor in the development of the disease. The average housewife does not soar beyond a plain bake, fry, or boil, and rich, highly-seasoned food is quite absent from the dietary of the class we are considering. When the finances are flourishing, no doubt, large amounts of animal food are consumed, but I have never met with gout in a non-beer drinker however large an appetite he might have, while, as I have before remarked, the really poverty-stricken classes do not develop gout.

ACUTE ATTACKS.

The acute attack presents no distinctive features from those exhibited by the patient of any other grade of society. There is the same excruciating pain, accompanied by the familiar polished, shining, tightly-stretched, thin-looking skin, with enlarged veins, and the surrounding area of œdema. The temperature rarely exceeds 100° F.; the tongue is thickly coated, the breath fœtid, and the bowels constipated. In the great majority of cases the urine is scanty and concentrated, throws down urates when cold, and contains albumen which may vary from a slight trace to a very considerable cloud. Casts of tubules are not met with during the early stages. The severity of the symptoms abates immediately upon treatment.

Pain has disappeared in five or six days, and in two or three days more all signs of inflammation have subsided. The metatarso-phalangeal joint of the great toe is, with rare exceptions, the first joint to be affected. Very occasionally a patient will tell you the whole foot was first attacked, but I have not met with any cases where the knees or joints of the upper extremity were the primary seat of disease. The joints which are most frequently attacked are the great toes, small joints of the foot, ankle, wrist, fingers, elbows, knees, hips and shoulders rarely, lumbar vetebræ very rarely. In some cases, although the first manifestations of the disease have appeared in the feet, all further attacks are confined to the wrists and fingers.

CHRONIC GOUT.

The cases, to which the term chronic gout is applied, are those which a long series of acute attacks has left in a permanently crippled, maimed, and deformed condition. These unfortunate persons may have every joint practically immovable, with enormous deposits of urate of soda deposited in and around the joint, distorting its shape and limiting or entirely preventing movement. The interphalangeal joints present a fusiform appearance, the deposit gradually tapering off. The swellings are not by any means regular, as nodular masses of the urate of soda cause the covering skin to become very thin, shiny, and blue, and often to give way altogether, permitting the escape of either a thick milky fluid, or of a portion of a dry chalk-stone. The hamstring muscles of the posterior part of the thigh become permanently contracted and shortened, and cause the knees to be fixed in a more or less flexed position. Considerable deformity and limitation of movement also occur in the wrists, elbows, and ankles. The ulceration of the skin over a chalk-stone leaves a very indolent ulcer which may persistently refuse to heal. The prepatellar and olecranon bursæ, and that over the metatarso-phalangeal joints of the great toe, are most frequently affected by the deposit of urate of soda. The formation of tophi in the cartilages of the ears is by no means universal, many cases in which chalk-stones are abundant about the joints failing to show a deposit in this situation. All cases do not, however, present the formation of chalk-stones ; and joints may become much distorted and absolutely stiff without the visible presence of any urate of soda.

A very characteristic deformity of the fingers which I have not hitherto seen described is caused by the hyper-extension of the joint formed by the proximal and middle phalanges, and by the flexion of the joints between the middle and distal phalanges. The bones are practically fixed in these positions, the joints permitting very little play. In some of these cases, although the cause is undoubtedly gout, the fingers assume a thin tapering shape due to the disappearance of subcutaneous fat, while the skin becomes thin, smooth, pink and glossy, suggesting the existence of some trophic condition. In all these cases there is a singular freedom from pain ; and in the absence of an acute attack the various joints perform comfortably such actions as their limited powers of movement permit. Deformities of joints once produced appear to be permanent. I have never seen a stiff joint regain its movement, nor have I known deposits of urate of soda to be absorbed.

The wrists and ankles in so-called chronic gout suggest to the touch that they are encased in a subcutaneous coating of plaster of Paris, and this is particularly the case when there are no perceptible tophi in those parts. I have in the Infirmary at the present time a man, aged 50, who developed his first attack of gout nine years ago. He was admitted for treatment then, and he has returned at intervals of three or four months ever since.

On his first admission, although looking beer-sodden, he was a well set-up, active man. He is now quite incapable of acting as an ostler at a public-house stable-yard, and is very much crippled. His feet are much inverted, and he has very little movement in his ankles or in any joint of his feet. He hobbles about on the outside edge of his feet, with semiflexed knees and stiffened hips.

His fingers are incapable of being extended in any joint beyond a point midway between flexion and extension, but they can be flexed to a slight extent. His wrists are immobile, and feel as if cased in some hard substance ; his elbows are limited as to extension and supination. Yet the movements of which he is capable give him, in his intervals of freedom from acute attacks, no pain, and he is able to get through a fair amount of light work. His craving for stimulants of some sort has not abated; for having been told off to clean out a store, he managed to get hold of a bottle of rectified spirit, and was discovered in a condition of helpless drunkenness. On recovery he denied the existence of even a headache, and said he preferred the beverage to public-house whiskey. He has not been given a further opportunity of comparing their relative effects. His urine, which has always been albuminous in an acute attack, now shows a distinct cloud permanently. This condition is pretty constant in persons who have had a large number of acute attacks; and unless they succumb to some other complication, they run the usual course of cardiac failure secondary to kidney-disease, as shown by the early stage of increased blood-tension, tortuous thick-walled arteries, and hypertrophy of the left ventricle, and then a gradual wearing out of the heart, with dropsy, water-logged lungs, and finally death from sheer heart-failure or from uræmic convulsions and coma. At no time does the urine present the pronounced characteristics of the contracted granular kidney. Sugar is frequently present during an acute attack, particularly if the attack is the result of a prolonged drinking-bout. It may become permanent during the later stages of the disease.

In response to a request made by a physician to one of the London hospitals, I examined the joints of many patients dying from chronic kidney-disease: a very considerable proportion showed crystals of urate of soda in the joints, although none had given a history of antecedent gout. I was quite unable to determine why some cases showed the deposit and others did not. The point is a very interesting one and worthy of further study.

I have often noticed that persons dying of wasting diseases, especially those suffering from cancer or phthisis, develop an acute inflammation of one or more joints. The ankle is most commonly attacked, sometimes the wrist, and seldom the elbow. In every case in which I have had an opportunity of making a post-mortem examination urate of soda was found on the cartilages and in the synovial fluid of the affected joints.

There was no previous history of gout in any case.

IRREGULAR GOUT.

My experience of irregular gout, *i.e.*, manifestations attributable to gout appearing elsewhere than in a joint, is very limited.

Dyspepsia and gastro-intestinal catarrh are common, and are usually to be traced to an excess of drink or to dietetic errors. They can be readily cured by suitable dieting, and do not respond particularly to an anti-gouty treatment.

Chronic bronchitis is the most common form of trouble associated with gout. It is never synchronous with an attack of articular gout, nor is it influenced by the administration of colchicum, but it invariably and rapidly disappears upon the occurrence of an articular attack.

Palpitation of the heart is not infrequent. It is usually associated with a flatulent distension of the stomach and intestines, and readily responds to treatment directed to this condition.

Eczema is undoubtedly a frequent manifestation of gout, and it may either be general or local; if the latter, then the site is determined by some special irritation. The lower third of the leg is especially prone to the affection, particularly if the veins are inclined to be varicose. In this condition the administration of alkalies and colchicum certainly appears to cut short the affection. The eczematous condition may sometimes develop into a chronic ulcer of the leg; and several patients who have suffered from gout, and have subsequently developed an ulcer of the leg, have informed me that so long as the ulcer is "open" they do not get an attack of gout or of bronchitis, but that when the ulcer heals they invariably get either one or the other. This is a widely-spread popular idea, and I was at first inclined to treat it with scant respect; but finding that when, in spite of their protests, by enforced rest in bed and constant fomenting their ulcers were healed, the expected attack of gout or bronchitis in due course actually appeared, I have treated the observations of patients with more deference, especially as I have on more than one occasion seen the healing of a long-existing ulcus cruris followed by cerebral hæmorrhage, this sequence being also the foundation of a widely-spread popular belief.

I was once much puzzled by the condition of a patient who was admitted suffering from acute gout and also presenting areas of sloughy superficial inflammation over the back, neck, buttocks. and the posterior parts of the thighs and calves. The man could give no satisfactory account of his condition but his clothes were very damp, and were redolent of sulphuretted hydrogen, and it appeared that, having become helplessly drunk one evening, he had laid himself down upon a heap of "Blue Billy," which is a popular term for the refuse lime which is cast out of the refining chambers of a gas-house, and which contains much sulphide of lime. It rained during the night, and the very irritating character of his couch resulted in some extensive burns, which, however, healed very kindly.

Gouty persons also appear to be specially liable to attacks of *phlebitis* in the superficial varicose veins of the legs, but the condition presents no departure from the usual course as presented by non-gouty patients.

Muscular pains and tingling in the hands and feet are often complained of, and are usually the result of "kicking over the traces" in the way of taking a glass or two of the forbidden liquor.

Depression of spirits occurring on the termination of a debauch is common, and is I think more pronounced in persons of a gouty character. The old-fashioned, but effective, blue pill and black draught usually removes the black cloud of care and puts a more roseate hue upon the affairs of life.

A condition of the bladder almost amounting to *strangury* is occasionally observed during the height of an acute attack. It appears to be caused by the passage of a very highly concentrated albuminous urine and rapidly disappears upon the dilution of the urine by the taking of large quantities of bland fluid. I have met with patients who described a well-marked *urethritis* as being gouty, but as my knowledge of their habits enabled me to form the opinion that they had "made a bid" for the ordinary sort of gonorrhœa, I have up to the present seen no reason to suppose that such a condition as gouty urethritis exists.

I have no experience whatever of *retrocedent gout,* possibly because "institution" patients and inmates lead such very methodical lives and are so carefully looked after that they get very little chance of indulging a capricious appetite or of applying any irregular or unorthodox treatment to an affected joint.

Parotitis certainly occurs in debilitated gouty patients, and sometimes progresses to the formation of an abscess. *Post-mortem*, the gland is sometimes found to be riddled with small discrete collections of pus. As a matter of clinical interest, metastatic swellings of the parotid are most frequently seen in debilitated sufferers from chronic joint-troubles, and in persons suffering from any form of chronic ulceration of the cæcum.

A severe form of *stomatitis* and *gingivitis* is common in gouty subjects.

DIAGNOSIS.

The only joint-condition which is at all likely to be confused with gout is that presented by *osteo-arthritis*. Patients suffering from the latter disease are subject to a very severe form of acute attack affecting chiefly the ankle-joints and the joints of the small bones of the feet, the wrist-joint, the elbow, and less often the knee. In these attacks the joints and the tissues around are swollen, red and exceedingly painful to touch and to movement. The inflammation is not quite so intense, nor is the skin so tense and shiny as one is accustomed to see in an acute attack of gout, but there is a real difficulty in determining between an acute attack of osteo-arthritis and a subacute attack of gout. The presence of tophi naturally helps when one can find them, but they are often absent even in well-marked cases of gout.

The most extreme cases of osteo-arthritis gravitate towards the Poor Law Infirmary, and I have had cases under my care in which the only movement possible was that of opening and shutting the eyelids, every joint being absolutely immovable.

The main fact to be kept in mind is that in gout the deformity is in the fibrous structures *around* the joints, and that there is little or no actual alteration in the shape of the surfaces of the joints themselves; whereas in osteo-arthritis it is the joint-surfaces and the bone in the immediate neighbourhood which cause the deformity and consequent limitation of movement. As I have before pointed out, the ulnar deviation of the fingers is not absolutely characteristic of osteo-arthritis; it is probably an "occupation" deformity, and does sometimes occur in well-marked gout. The history and mode of onset is the safest guide in coming to a correct diagnosis.

Neither *gonorrhœal* nor *syphilitic* synovitis should cause any great confusion. The large amount of synovial fluid in the former case, together with the absence of periarticular heat and redness, sufficiently distinguishes it; whilst the latter is only likely to be confused with tubercular synovitis.

Gout and osteo-arthritis do, however, occur in the same person. I have since the commencement of this paper made an autopsy upon a patient who died from the effects of chronic interstitial nephritis. He first came under my care about eight years ago, suffering from gout affecting the great toes, ankles, wrists, elbows and knees. The attacks were well marked and the urine was albuminous throughout. Three years ago he became a permanent inmate because of a very stubborn attack of chronic eczema. He had had no attacks of gout since his last admission, and although his eczema was eventually cured, the kidney-condition and the consequent train of symptoms dependent upon cardiac failure caused his death within the past few days. All the joints were carefully examined and they all showed signs of the fibrillated plush-like condition of the cartilaginous surfaces of the joints, together with commencing disappearance of the cartilage in patches and some erosion of it at the edges of the joints. The great-toe joints were, however, the only places where any evidence of gout was found, and here the ligaments and tendinous expansions were infiltrated with deposit, and there were some small patches of urate of soda in the cartilage of the proximal end of the proximal phalanx of each great toe. The kidneys were typically granular and contracted, with numerous retention-cysts; and the left ventricle of the heart was greatly hypertrophied. There was a generally water-logged condition of the body.

Here then was a case which showed very little articular sign of the large number of acute attacks of gout from which he had suffered, but which gave definite evidence of the presence of osteo-arthritis, the existence of which was unsuspected during life.

As before remarked, the tapering of the fingers, due to the absorption of subcutaneous fat, and the smooth, glossy condition of the skin of the fingers, which suggests a trophic origin of the condition, occur in gout as well as in osteo-arthritis.

MORBID ANATOMY.

I have not had an opportunity of making a post-mortem examination upon a case of acute gout, but in long-standing cases of the disease the cartilaginous surfaces of the joints appear as if they had been whitewashed; the deposit is thickest at the central part of the articular surface, and gradually fades away towards the periphery of the joint, there being always a marginal zone which is free from deposit. The synovial fluid is milky from suspended crystals of urate of soda. The ligaments, tendons, and tendon-sheaths, in fact all the fibrous structures around a joint, may be thickly infiltrated with the deposit, which may form hard, dry, chalky masses, or may occur in fluid milky collections in the vicinity of a joint. True exostoses undoubtedly occur, and I have often seen the great-toe joints surrounded by a true bony outgrowth. The extreme distortion shown by the joints and the semi-luxations which occur are produced by extra-articular causes, the actual surfaces of the joints showing little departure from the normal shape, although the margins of the articular surfaces, particularly of the larger joints, may show considerable lipping from coincident osteo-arthritis. True fibrous ankylosis does not occur. Although I have carefully looked for them, I have not found any deposits in the kidneys.

Arterio-sclerosis and chronic interstitial nephritis are constantly found in cases of gout which have lasted over ten years; indeed, uræmia is, perhaps, the most common cause of death in gouty subjects. The presence of albuminuria in such a large percentage of cases in the early stage of the disease suggests that the arterio-sclerosis and coincident hypertrophy of the left side of the heart are secondary to the kidney-condition, which in cases of long duration is of the most pronounced chronic contracted granular variety.

TREATMENT.

From the dietetic side of treatment, institution life has the very great advantage that, within a little, one can be certain that there is no kicking over the traces. Until all acute symptoms have subsided, I insist upon an exclusive milk-diet. No limit is placed upon the amount, and frequently five or six pints daily are consumed. On the subsidence of the attack, bread-and-butter is allowed for breakfast, tea, and supper, with rice- or sago-pudding for the midday meal. After a week or so, fish and potatoes are added to the dinner, and after another few days the patient gets the full "No. 1" diet, which consists of practically an unlimited amount of bread-and-butter and tea twice daily; a midday dinner of 4 ounces of cooked meat, 8 ounces of potatoes, 4 ounces of rice- or sago-pudding, with cabbage or other green food twice weekly; and a supper of bread-and-milk, which completes the dietary. A relapse almost invariably occurs when, in response to repeated and sometimes exceedingly impolite demands for a change of food, a too early return to meat or even fish is given. Alcohol in any form is absolutely withheld, and I repeat emphatically my firm conviction that malt-liquor is the cause of the gout amongst the so-called poor, and also that, however advanced the disease may be, when once the patients have settled down in the infirm wards of an institution where malt-liquors are withheld, no further attacks occur.

Whilst on the subject of diet, I should like to draw attention to the prevalence of true scurvy amongst the tramp class, which I mentioned in a communication to the *Lancet* in 1887. Since that time I have seen many cases, all showing hemorrhages into the tissues of the popliteal space, thigh, and calves, and associated with sponginess and marked swelling of the gums. These confirmed "casuals" live upon a diet of bread and cheese and oatmeal for months together; meat and vegetables are unknown to them unless they can "cadge" a meal, whilst any coppers derived from the eleemosynary charity of the public are spent in procuring the temporary oblivion from wretchedness produced by public-house alcohol.

Medicinal Treatment.—The administration of 5 grains of calomel and an ounce of "house-mixture" forms the invariable preliminary treatment upon the admission of a case of acute gout. Our stock Colchicum-mixture consists of ♏. xv of colchicum wine, gr. x of Mag. Carb. Pond., gr. xl of Mag. Sulp. ♏. x of Tincture of Hyoscyamus, ♏ x of Spiritus Ætheris Nitrosi with Aq. Menth. Pip. to ℥ j. This is usually given every four hours until the acute symptoms show sign of abatement, and then the amount is reduced by giving it only three times daily. The continued administration of colchicum is stated by some authorities to be harmful, but my experience does not confirm this. I have met with cases where, through an oversight, the patient has not been "taken off" his medicine upon the cessation of the acute symptoms, and where the zealous sick-ward attendant of the workhouse has diligently given the mixture three times a day for months without any untoward symptoms supervening. Upon the introduction of Piperazin I gave it an extended and careful trial, but as I could get no results whatever from its administration I abandoned it.

Salicylate of soda does not appear to have any appreciable effect in either diminishing the pain or cutting short the duration of an acute attack. The articular pain and stiffness sometimes complained of by the sufferers from joints deformed by gout and by coëxistent osteo-arthritis are relieved by a mixture of the confections of sulphur and of guaiacum, and also by the giving of five-drop doses of the old-fashioned balsam of sulphur with an equal amount of turpentine. The most convenient way of taking these drops is upon a piece of loaf-sugar.

The only local application which I have found of use is a hot-brine fomentation which we make from the common rock-salt: this is really of great value. I gave the local hot-air treatment a trial lasting for 12 months, but beyond consuming a fearful quantity of gas I cannot say that I achieved any notable result.

ON THE RELATION OF GOUT TO GRANULAR KIDNEY AND TO LEAD POISONING.

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EXCESS of uric acid in the blood, or *Uratæmia* as Sir William Roberts named it, we are bound, so far as our present knowledge goes, to consider as the essential factor in gout. That being granted, enquiry into the cause of gout involves the study of the natural history of uric acid in the body and the conditions which determine its excess in the blood.

Given the essential condition, viz., uratæmia, it is necessary next to study the conditions which determine the deposit from the surcharged blood, of uric acid or urates, either within the body as in the joints, or external to it in the urine or urinary passages.

What has just been written would seem to imply a close relation between gout and uric-acid gravel or calculus. Yet the connection between them is by no means so close or simple as might be supposed, for urine may contain excess of uric acid without deposit, and may deposit much when the amount is not large. In cases of uric-acid calculus and gravel a history of previous gout is rare, and in places where stone is common, as in Scotland or in Norfolk, gout is rare; while in India among the natives uric-acid gravel and calculus are frequent, but gout unknown.

Deposits of uric acid are not unfrequently seen *post mortem* in the kidney as minute nodules in the cortex, and as streaks in the pyramids, but these have clearly no relation with gout, and they are commonest in children who have died of very various affections. In adults they are very rare in fatal cases of gout, and are more frequently found where the kidneys are otherwise sound and where there is no other evidence of gout, clinical or pathological. Thus it appears as if the conditions leading to gout and uric-acid gravel or calculus respectively were more or less antagonistic.

Given the deposit, many of the results are the more or less mechanical and necessary consequences of the presence of a foreign body such as the deposit of uric acid or urates becomes. In the joints the deposition leads to the inflammatory and other symptoms characteristic of acute gout, and subsequently to the more permanent changes associated with chronic gout. So too in the kidney—assuming the connection, which I have already said does not seem really to exist. between uric-acid calculus or gravel and gout—many of the changes of the so-called gouty kidney are the pathological consequences of a calculus, irrespective of its nature.

The acute attack of gout is associated with certain changes in the urine. The urine is concentrated, strongly acid, of high specific gravity, and throws down on standing a copious deposit of lithates. These changes have little to do with gout itself, but are to be referred to the general dyspeptic and constitutional disturbance by which the acute attack of gout is attended, for urine with the same characters is passed in many other disorders, especially those of a febrile character, *e.g.*, rheumatic fever and pneumonia.

As in these conditions, so in acute gout, the urine may temporarily contain albumen, usually as a trace only or, at any rate, in small amount. Sometimes, however, it is present in considerable quantity. Thus I have seen specimens in which, on boiling, it was found to be a half or more, and in one case the urine became solid. Yet in all these instances alike the albuminuria was transitory, lasting only during the acute stage, and after a few days disappearing entirely.

The occurrence of albuminuria during acute gout has been referred to irritation set up in the kidney by the passage of the acid and concentrated urine, but the irritation does not, at any rate, lead to acute inflammation, for the association of acute nephritis with gout is extremely rare, and patients suffering from acute nephritis do not develop symptoms in any way resembling those of gout. If a gouty patient develop acute nephritis, it is either an accidental and independent affection, or an acute attack resulting from previously damaged kidneys, in other words from antecedent interstitial nephritis.

The repeated irritation of the kidneys set up by repeated attacks of gout might lead to degenerative changes of a permanent kind, and this is the explanation often given of the association of long-standing gout with chronic interstitial nephritis.

"Gouty kidney" is a very unscientific and misleading term, for the gouty nature of it is often a matter of personal opinion only. The gouty kidney, as often described, presents a mixture of fatty degeneration with interstitial change, and may have nothing whatever to do with gout. Thus if the presence of a uric-acid calculus or gravel be considered proof of gout, the urine might include the effects of this calculus upon the kidney, and would thus embrace many of the conditions often described by the term "surgical kidney"—another equally indefinite and misleading expression. But if it be true that there is not only no relation between gout and uric-acid calculus or gravel, but rather an antagonism, all the resulting pathological conditions must be excluded.

In other cases the lesions of the so-called gouty kidney are due to an atheromatous condition of the vessels, the fatty degeneration of the cells being the result of the imperfect circulation. There is no direct relation between gout and atheroma, except that chronic gout is met with usually in elderly persons. Similar conditions of the kidney may be met with in old persons with atheromatous vessels who have never had any manifestation of gout in their whole life.

It now remains to consider the relation which exists between gout and chronic interstitial nephritis or granular kidney.

The association of gout and granular kidney is a close one, and appears clearly as well in the statistics of gout as in those of granular kidney, for many gouty patients are found to have granular kidneys, and among cases of granular kidney gout is a very common complication. Thus Ord and Greenfield found that of hospital patients who had uratic deposits in the great-toe-joint two-thirds had granular kidney, and in the remaining third the kidneys were not sound. Norman Moore, in 49 cases of granular kidney, found uratic deposits in 22. Pye-Smith, in 10 cases of fatal gout, found that every one had interstitial nephritis.

On the other hand, granular kidney and gout are not necessarily associated. Thus, in 69 cases of granular kidney, Dickinson obtained distinct evidence of gout only in 16. And, again, Norman Moore's figures show a marked difference between the sexes, for of 26 cases of granular kidney in women only five showed uratic deposits, or less than half the relative number in men.

Further, many persons have gout all their lives, and yet the kidneys are found healthy at the end, or show changes which are not connected with atheromatous vessels, the atheroma being due to age rather than to gout. As stated, gout does not produce atheroma at all, or at the most only aggravates or intensifies the natural tendencies of age.

If then, on the one hand, some patients may have gout and never develop granular kidney, and on the other some may have granular kidney and never gout, however close the relation may be, it is not constant or essential.

Gout and granular kidney are both of them very common affections, and sufficiently common to be not infrequently associated accidentally without any causal connection. Still, making allowance for this, the association seems altogether more common than mere coincidence could account for.

It is difficult to discuss satisfactorily the relation of two conditions to each other, when neither condition admits of precise definition, for some authorities are more easily satisfied in the diagnosis of gout than others, and while some place all forms of chronic interstitial nephritis in one and the same category, others are not so comprehensive, and regard granular kidney as a definite clinical disease, of which the interstitial nephritis is only a part.

Accepting for the present purpose the association of gout and granular kidney as established, the question of the relation in which they stand to one another still remains to be dealt with. Does gout cause granular kidney or does granular kidney lead to gout, or is there some less direct relation between them?

Gout is a disease of the later half of life, lasts many years, and is rarely fatal of itself. Granular kidney is a disease of earlier life, and terminates at a much earlier average age, and the general clinical history of gout is not that of granular kidney.

The relation of lead-poisoning to gout will throw some light upon the question now under consideration.

The association between both these conditions and granular kidney is very close. Experimentally, lead-poisoning has been shown to cause in acute cases epithelial degeneration in the kidneys, and in chronic cases, interstitial change in addition; in these respects resembling what has been described in gout. In many cases of lead-poisoning the kidneys are found to be granular. This is especially true of the fatal cases, and, though *post-mortem* figures may not prove that lead produces granular kidney, it clearly establishes the fact that the association of the two conditions is very grave.

Between lead-poisoning and gout there is also a close relation. This Garrod's original observations established, and they have been amply confirmed by other observers. Garrod, in 100 cases of gout, found 33 cases of lead-poisoning, and Duckworth 25 out of 136. Yet the association is by no means so close as these figures appear to prove. Thus Oliver has pointed out that, among the lead-workers in the district of Newcastle, gout is rare, but that, though the native lead-workers rarely develop gout, the workmen who go there from the south do—a difference which is probably to be explained by a difference in the habits of the two classes, especially in respect of drink. Again, in Berlin, Frerichs did not find in 163 cases of lead-poisoning a single instance of gout.

Where chronic lead-poisoning and gout are associated with granular kidney, the patients are very cachectic, but this cachexia may as well be the result of granular kidney as of lead.

The conclusion is thus arrived at, that though chronic lead-poisoning may lead to some interstitial change in the kidney, it does not of itself produce granular kidney.

Another conclusion may also be drawn, viz., that patients whose kidneys are unsound are especially liable to suffer from the effects of lead, and that the association of the two conditions may easily lead to serious or even fatal results.

These conclusions are much the same as those to which the considerations of the relation between gout and granular kidney have already led. It may in both cases be explained as the result of renal inadequacy—the elimination, in the one case, of the lead, and in the other of the uric acid being interfered with; while it is possible that lead, by modifying the general metabolism of the body, may directly affect the production or elimination of uric acid, and thus lead to the excess in the blood which is the essence of gout.

We thus arrive at much the same conclusion in respect of both gout and lead in relation to granular kidney, viz., that, though each may produce chronic changes in the kidney, neither causes granular kidney. But the presence of granular kidney greatly increases the liability of the patient to gout on the one hand, and lead-poisoning on the other, or to both together, and in each affection alike greatly increases the gravity and risk.

THE CAUSATION, PREVENTION, AND TREATMENT OF GOUT.

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GOUT is the old name of that form of arthritis caused by uric acid which commonly affects people in middle life, and is more often, at least at first, to be found in a single joint. Rheumatism is the old name of the arthritis produced by uric acid which commonly affects young people and many joints at a time. Arthritis due to uric acid includes both of these main forms and all intermediate forms between them which are to be found in nature, and it is impossible in these days to write about the arthritis which used to be called gout without also writing about the arthritis which used to be called rheumatism. Further knowledge shows us to-day that we have to do with but one disease, irritation of fibrous tissues in and about joints and elsewhere due to uric acid. This we can now see is not, strictly speaking, a disease at all, but a reaction of the physiology of the body to food-poisons; and if the food-poisons are left out, the reaction after a time fails to appear.

Gout is not a constitutional disease due to any defect in the formation or functioning of the body. It is a form of "diet" disease due to food-poisoning. It follows from this that it can be cured by leaving off the poisonous foods, and prevented by omitting such foods from the diet of the young.

CAUSATION.

When the possibility of introducing uric acid into the body and blood by swallowing it like other poisons was neither understood nor believed in, it was naturally concluded that gout was not due to poisoning, but was a constitutional defect or disease. I believe I was the first to point out in a paper in the *Journal of Physiology*, Vol. XV., published in 1893, that the experiments on which these erroneous beliefs were founded had serious flaws in them—flaws which were due to the generally-prevailing ignorance of many of the conditions that control the solubility of uric acid in the blood, and its excretion in the urine.

But when once these conditions had been followed a little more closely and in experiments extending over considerable periods of time, it soon became evident that one had only to administer a few grains of uric acid, while taking care to keep the blood in a condition to dissolve them and hold them in solution, to find them appearing with the greatest punctuality in the urine, grain for grain as administered.

And this was the great mistake that the older experimenters made; they administered 20 or 30 grains of uric acid, paying no attention to its solution in the blood, the result being that it was all retained in the liver and only got into the blood and urine some days later, after the experiment was over. The uric acid thus swallowed went into the stomach and was absorbed from the intestinal tract into the blood. It then passed by the portal circulation into the liver and was retained. But it was not only retained, it formed in the liver a "uric-acid filter,"[1] which attracted to itself all the uric acid that came past it from the intestines or that which was otherwise floating in the blood. Hence the blood was cleared of uric acid and the excretion of uric acid in the urine was diminished. But clearing the blood of uric acid increased the formation of urea (*see* "First Principles in Uric Acid," *op. cit.*, Ch. IV., especially page 153). The old experimenters found that the administration of uric acid did not increase the excretion of uric acid, but that it did increase the excretion of urea; and they then quite unwarrantably jumped to the conclusion that the urea which came out was the product of the uric acid which went in, a conclusion which was quite in accord with old physiology, but not at all in accord with modern investigations.

We now know that if the uric acid had been swallowed after a course of alkali, which would have prevented its retention in the liver and its production there of a "uric-acid filter," it would have remained in solution in the blood and have passed out in the urine almost as quickly as it was swallowed.

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[1] *See* "Uric Acid Filter Experiments" in *Uric Acid*, 6th edition, page 177.

It was all along a mere question of solubility in the liver and blood, about which, however, the older experimenters knew nothing. They concluded that the uric acid swallowed was oxidised in the body into urea, and that "gout" and "goutiness" were due to failure of this oxidation. As a matter of fact uric acid was never oxidised in the one case or in the other. When given without solvents, it simply remained in the liver until the next time the supply of alkali in the blood favoured its solution; and it then began to pass into the blood and urine. When given with solvents, it passes into the blood and urine grain for grain as swallowed, so that the excretion can be made anything from 10 grains a day to 30 grains a day, according to the amount swallowed. Then as the introduction of most meat-takers and tea-drinkers is from 8 to 10 grains a day, or nearly 8 to 10 ounces in a year, we can easily see that there is here a huge amount of uric acid passed into the body sufficient to account for almost any series of troubles that can be produced by this poison. Those who are constantly passing all their uric acid through their blood suffer from the great "collæmic group" of uric-acid diseases, and those who, from the defective solvent power of their blood, retain it in the fibrous tissues suffer from the irritation of these fibrous tissues which we have been accustomed to call gout and rheumatism;[1] and any fibrous tissue in the body may be affected.

Now solubility in the blood depends practically, as we see in "uric-acid filter" experiments, on the amount of alkali in the blood, and those will have most alkali in the blood who have eaten most vegetables and potatoes, who clothe warmly or live in warm climates or take much exercise, and who are frequently hot, but who do not overfeed, as the more the food the higher the urea, the higher the acidity of the urine, and the lower the alkalinity of the blood. Those on the other hand have least alkali in the blood who live in cold climates, who under-clothe and overfeed, and who may further take acid beverages such as beer or wine, who eat few vegetables and take little exercise. Hence Cullen's observation quoted in *Uric Acid* (page 864), "that gout seldom attacked persons employed in constant bodily labour, or those who live much upon vegetable diet." Hence also Sydenham's observation on himself that

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[1] For a classification of these two groups of diseases see *Uric Acid*, p. 135.

"whenever I returned to my studies gout returned to me (*op. cit.*, page 729).

Those that retain the uric acid they swallow will have gout in one form or another; those who excrete it as they swallow it will have collæmia, anæmia, Bright's disease, and other members of the great collæmic group. *No one swallows uric acid with impunity, but freedom from all troubles can easily be obtained by refusing to swallow it.* In the same way solubility explains completely the periods of life at which excess of uric acid produces its chief effects. Thus with high-nutrition periods we get the retention-troubles which we used to call gout and rheumatism, and in low-nutrition periods we get collæmic diseases, or what are sometimes called "gout of the blood."

Now life may be divided into four such periods, two of high nutrition with retention of uric acid, and two of low nutrition with excessive excretion of uric acid and its passage through the blood in excess. High nutrition means large metabolism, high acidity of urine, diminished alkalinity of the blood; and low nutrition means the reverse of all these conditions. Now the two high-nutrition periods of life are (1) that of growth and rapid metabolism up to 14 or 15 in girls, 17 or 18 in boys, and (2) that of middle life with great vigour and endurance, and high development of muscle-power. The two low-nutrition periods are (1) from 14 to 25 in girls, when there is a slackening of the metabolism after the great developmental increase, and from 17 to 25 in boys, the condition being similar to that in girls but rather less marked; and (2) the low-nutrition period of the decline of life from 50 onwards in both sexes, according to the strength of the individual. The retention-disease of the first high-nutrition period is that which we have been accustomed to call rheumatism, and the retention-disease of the second high-nutrition period of middle life is that which we have been accustomed to call gout. Both we can now see are simply arthritis due to uric-acid poisoning. The collæmic diseases of the first low-nutrition period are chlorosis, anæmia, headache, epilepsy, hysteria, Bright's disease, and others; and the collæmic diseases of the last or later-life low-nutrition period are anæmia, pernicious anæmia, melancholia, Bright's disease, diabetes, and obesity (these last three being the defective combustions of albumen, sugar, and fat produced by defective circulation).

Some may wonder why in a paper on gout I should mention all these diseases, but I should simply have to reply that it is impossible to understand some effects of a diet-poison without also understanding as far as possible all its effects, and the relation of one series of effects to the other. The exact relation of the retention-trouble, "rheumatism". to the collæmic disease "chlorosis" which follows it, is now, I hope, quite simple and refers merely to time of life.[1] If uric acid was not swallowed, neither of these forms of uric-acid poisoning would ever be seen. Further, I will say that no drug that ever did good, or that will ever do good, in the arthritis which we have been accustomed to call gout or rheumatism has any action but that of a solvent of uric acid, and the powerful salicylates are useless or even harmful in these troubles if their solvent action is prevented.[2]

To-day we can produce all forms of arthritis and other so-called diseases simply by swallowing uric acid. But we can do much more than this. We can actually watch the passage of this uric acid through the blood, and so map out the hours at which it goes through. For it produces in passing quite decisive changes in capillary circulation.[3] This knowledge then raises two questions: (1) How much uric acid is introduced into the body in the food of an ordinary day? and (2) Will the uric acid thus introduced suffice to account for all the uric acid of disease? The answer to the first question has already been given.

The answer to the second question is that these quantities if all retained in the body will mount up and form 8 to 10 ozs. of uric acid in the year; and though under ordinary circumstances nothing like the whole of this amount is retained in the body, such an extensive introduction going on year after year for 30 or 40 years with even quite a small retention, will easily furnish all the uric acid that we ever see inside a human body.

One other fact which we have to bear carefully in mind is that *introduced uric acid* interferes with the solubility, and so with the excretion, of the uric acid that is formed in the body. For every one forms uric acid every day in about the relation of 35 grains of urea and 1 grain of uric acid for each 10 pounds of body-weight, and the man who suffers from gout probably *forms* no more uric acid in relation to urea than any one else does.

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[1] See *Uric Acid*, Fig. 59.

[2] See *Uric Acid*, pages 793 and 797, *seq*.

[3] See *Uric Acid*, Figs. 74 and 75.

On the other hand, if the reverse were true, if the gouty man from some defect in structure or function formed more uric acid, either relatively or absolutely, than anyone else, this would go on in spite of diet and drug-treatment, and it would be impossible either to prevent or cure gout or other uric-acid diseases.

As a result of this effect of introduced uric acid on the solubility of uric acid in the blood, a man who forms 10 grains of uric acid a day and introduces other 8 or 10 grains each day in his food, will probably fail to excrete all the uric acid he has to deal with, and will soon accumulate a more or less large store in his body. On the other hand, the man who forms 10 grains a day, but only introduces 1 grain a day, may very probably excrete day by day all the uric acid he has to deal with, and may consequently never form a store or accumulation at all. In other words, all the uric acid of *formation* does no harm, provided that a large amount of *introduction* is not added on to it.

I must guard against misunderstanding by saying that the above is only true where no more than the physiological allowance of albumen is taken (*i.e.*, that which suffices to produce from 3 to 3·5 grains of urea per pound of body-weight per day). But if this quantity be exceeded, there will, of course, be an increased formation both of urea and uric acid, but still in the relation of 1 to 35; and, as a large nitrogenous metabolism means, for obvious reasons, a diminished alkalinity of the blood, some of the formed uric acid may be retained and produce an accumulation.

It is necessary, therefore, to look to two points :—(1.) To introduce into the body as little uric acid as possible; (2) not to take more than a physiological allowance of albumen. Probably a considerable number of those who are gouty to-day have for years been breaking both these rules, and their sufferings are the result of their doing so. Still, of the two, introduction is by far the larger and more important factor, so that we are justified in saying that gout in this country is due to poisoning by animal flesh and tea.

Speaking generally, the muscles of animals may be said to contain uric acid to the extent of from 5 to 8 grains to the pound, and their viscera (liver, kidneys, &c.) from 6 to 12 grains to the pound, and these quantities are introduced into the bodies of those who swallow them. Then the alkaloids of tea, coffee, and cocoa are, as is well known, xanthines, and these for physiological and pathological purposes are equivalent to uric acid; and several other vegetable substances, such as peas, beans and lentils, mushrooms, and asparagus, contain considerable quantities of xanthines, some of them even twice as much as meat. As regards the xanthines, I have shown that their administration produces just the same effects as the administration of uric acid.

We have thus seen where the uric acid comes from. We have now to see how it acts, and for this purpose we may divide its effects into two classes:—(1) The local irritation it produces in the joints and fibrous tissues (gout and rheumatism); and (2) the general symptoms, often called "gout in the blood," *i.e.*, the circulatory obstruction with high blood-pressure, and its other results, &c.

(1) The former question has always seemed to be fairly simple, for uric acid is an irritant of tissues especially when it is in solution; thus Ebstein showed years ago that uric acid in solution with phosphate of soda produced irritation of the cornea when injected into it, while the phosphate solution alone produced no irritation. Then Pfeiffer, of Wiesbaden, made some interesting experiments by injecting urates in suspension under the skin; and he found that when alkalies were taken the urates caused local irritation, but if acids were taken the irritation diminished—in other words, the urates in suspension caused little irritation, but when they were got into solution by the alkali the irritation was increased.

For my part I never found it necessary to make any experiment, for nature had already made quite sufficient experiment to convince me, in my own person, that uric acid was a local irritant, and I had long known that such irritation was made worse by precipitants and better by solvents.

Under some conditions also the use of alkalies may make the pains of arthritis worse—that is, when the alkali merely suffices to increase the solubility of the urate, but not to remove it from the focus of irritation. The more powerful solvents, the salicylates, relieve, not because they get the urates into solution, but because they remove them from the irritated joints and from the body.[1] On the other hand, we often see the biurate of soda lying deposited in various parts of the human body (toes, ears, &c.) without the smallest signs of irritation or inflammation round it. Hence we see that urates in solution are irritating, but urates deposited are not.

Another fact which points the same way is that the biurate of soda takes two to three days to be thrown down from its solution in serum, whereas wine will often produce an attack of gout in as many hours even before the patient leaves the dinner-table. Obviously, then, this attack of pain cannot be due to the throwing-down of the biurate, which has not had time to take place, but it must be due to a concentration ("uric-acid filter" action) of uric acid in the part, either in solution or in colloid form.

The biurate is a subsequent result of the gouty attack; and with the throwing-out of the biurate the irritation and inflammation subside, and we find it weeks, months, or years afterwards lying perfectly quiet in the tissues, its owner having possibly forgotten all about the attack of gout he once had.

In the blood at the time of the gout attack we have to deal with uric acid or urate containing more or less alkali, and either in solution or colloid form, like that which is thrown out from solution in phosphate of sodium on cooling or treating with a dilute acid. And it is the concentration of this urate, either in strong solution or colloid form, in certain fibrous tissues, that produces the local irritation and pain.

The reason it so generally selects the fibrous tissues is that these, being less vascular than many other tissues and less well supplied with alkaline blood, have greater difficulty in keeping up their alkalinity; and when this is diminished by any local cause, as cold and wet, or by a general cause, as the taking of acid wines or beer, the urate of the blood is rendered less soluble in these tissues, and is retained there probably in colloid form, producing more or less local irritation, according to the amount present in the tissues and the time during which it is able to act.

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[1] See *Uric Acid*, Fig. 62.

Then this retained uric acid constantly acts the part of a "uric-acid filter" and attracts other uric acid to it from the blood and tissue-fluids of the body.

The rule then about the action of drugs is this, that all substances that increase the power of the blood to hold uric acid in solution tend to remove it in solution from the joints and fibrous tissues, and to diminish the irritation in them because they remove it; while, conversely, all substances that diminish the power of the blood to hold uric acid in solution tend to cause its retention and accumulation in the joints and fibrous tissues, and directly or indirectly to increase the irritation in them.[1]

When we come to deal with the blood and circulation we shall see that all substances that increase solubility increase the amount of uric acid in the blood, while all substances that diminish solubility diminish the amount in the blood. We shall also see that the quantity in the urine always corresponds with the quantity in the blood, and that our power of control is so great that we can make the quantity in either fluid almost anything we like.

The chief substances that increase the solubility of uric acid in the blood are alkalies and salicylates; and heat has a similar effect, as it increases the passage of acids out of the body in perspiration, thus acting like a dose of alkali and increasing the alkalinity of the blood. These tend to prevent the irritation of fibrous tissues by uric acid, or to diminish such irritation when present by removing and excreting it. The chief substances that diminish the solubility of uric acid in the blood are the acids, and many metals that form insoluble compounds with uric acid; while cold naturally has the reverse effect to that of heat, preventing the excretion of acids in perspiration, and diminishing the alkalinity of the blood. Hence the arthritis of gout or rheumatism (for both are due to uric acid) may result from exposure to cold, or cold and wet, the taking of acid wines or beer, or contact either internally or externally with many of the common metals such as mercury, lead, zinc, and copper, &c.

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[1] *See* "Uric-Acid Filter Experiments," *Uric Acid*, p. 177.

I know of no point in connection with arthritis which these facts do not suffice to explain completely. That which looks most like an exception is met with in the case of the very chronic arthritis of debility and anæmia, often called rheumatoid. But here we shall always find excess of uric acid in the blood, and the joints always act as uric-acid filters and constantly take it up from the blood, thus increasing their own irritation. But if in this condition some uric acid is given, it clears the blood of uric acid, as we have already seen, and it therefore stops this irritation and the "uric-acid filter" action which produces it. Now a diet containing excess of meat acts in the same way as the administration of uric acid; it clears the blood of uric acid by effecting its retention in the liver. In the same way certain retentive drugs and waters all clear the blood of uric acid and hence stop the "uric-acid filter" action in the joints. There being no uric acid in the blood, the uric acid already present in the joints cannot attract more uric acid to itself and the local irritation subsides. Hence in such cases associated with anæmia and great debility, tonics, feeding up, and even meat-diet will do great good at times, and prevent the joint-irritation. The universal rule is that troubles made worse by solvents are relieved by retentives and *vice versâ*. The acute joint-diseases are relieved by solvents and made worse by retentives, the very chronic joint-diseases with great debility are relieved by retentives and made worse by solvents.

(2) The general symptoms of gout, often called "gout in the blood," depend chiefly on the fact that uric acid controls the capillary circulation of the whole body. It is probable that, being in solution with phosphates in the blood, it is under certain circumstances thrown out in the circulation as bulky colloid masses, more or less similar to those obtained when a solution of uric acid in phosphates is either cooled quickly or treated with dilute acids; and the control of the capillary circulation is probably due to the capillaries being obstructed by the colloid granules.[1] But be this as it may, the fact

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[1] For fuller treatment of these points I must refer to my work on *Uric Acid.*

remains that a careful measurement of the rate of the capillary circulation is an absolute guide to the amount of uric acid in the blood; for the capillary circulation not only varies from morning to evening in accordance with the physiological variations in the excretion of uric acid, and the amounts in the blood and urine, but it can be made to vary at any time and in any direction at pleasure by altering the quantity of uric acid in these fluids, which it is quite easy to do.

Perhaps a few definite figures will make this more easy to understand; and we have now also an absolute guide to the amount of uric acid in the blood in the number of granules precipitated in a drop of that fluid drawn from the end of the finger by the addition to it of a drop of a 10-per-cent. solution of sodii carb., followed by a drop of a 20-per-cent. solution of ammonium chloride. These granules bear a relation to the red cells, under physiological conditions, of, say, 1 to 8 in the morning, and 1 to 18 or 1 to 20 in the evening, there being much more uric acid in the blood and urine in the morning, as is well known. In pathological states they may be anything from 1 to 1 in Bright's disease, to 1 to 40 in fevers; and by the action of salicylates they may be made 1 to 5, 1 to 2, or even 1 to 1 in the first few hours of the drug's action, and they fall to 1 to 50 or 1 to 70 the day after it has been left off; corresponding exactly with the large fluctuations in the excretion of uric acid in the urine. In normal conditions it is possible by administering uric acid to make the day's excretion in the urine vary from 12 grains up to 30 grains, and the granules in the blood alter in the corresponding ratio.

Lastly, we must remember that obstructed capillaries in front, with a strong heart behind, involve of necessity high blood-pressure in the vessels between them; and this is the explanation of the uric-acid headache, to which I used to be a martyr, as in this disease the high blood-pressure causes undue pressure within the skull, and pain is the result.

I have called the return of the blood into capillaries blanched by pressure the "capillary reflux," and a careful measurement of the time this takes is the measure of the quantity of uric acid in the blood; and all the other phenomena—the number of granules in the blood and the blood-pressure—vary with this.

Thus the capillary reflux measured by my capillary dynamometer[1] and a metronome beating half-seconds is in the morning six or more half-seconds, in the evening three or four half-seconds, and we get the following groupings of events:—

—	Capillary Reflux.	Blood-pressure.	Granules in Blood.	Uric-Acid relation to Urea in Urine.
In health, Morning	6 half-secs.	120 mm. Hg.	1–8	1–18[*]
„ Evening	4 „	100 „ „	1–18	1–38[*]
Fevers - - -	2-3 „	70–80 „	1–40	1–50
Bright's Disease -	10–14 „	170–180 „	1–2	1–15

[*] Note.—The absolute quantity of uric acid is about 10–11 grs. per day, and with capillary reflux of 7–8 half-secs. from 18 to 20 grs. per day.

There is one other easily-observed result which agrees with those just mentioned in every way, and that is the urinary secretion, for obstructed capillaries in the skin and slow capillary reflux are the index of similarly obstructed capillaries throughout the body, and obstructed capillaries in the kidney mean that the water of the blood has difficulty in passing through the kidney into the urine. Hence it follows that the urinary flow under all conditions varies in the reverse direction to the uric acid excreted along with it. If the uric acid is rising the water is falling, and *vice versâ;* for excess of uric acid in the urine means, as we have seen, excess in the blood, and excess in the blood means obstructed capillaries throughout the body, and obstructed capillaries in the kidney mean scanty excretion of water from the blood.

Hence the urinary secretion is not only scanty in the morning and profuse in the evening, the reverse of the uric acid, but it follows every fluctuation in the excretion of this substance throughout life, whether due to physiology, drug-action, or pathology; and by controlling the uric acid we can also make the urinary water anything we please. It thus furnishes us with a ready index of what the uric acid is doing, and with one more easily-observable proof of the enormous power which this substance has in controlling the circulation, and thereby the physiology and pathology of the body throughout life. And, as I have said already, by means of drugs or by administering uric acid, we can produce results similar to almost any of the above at pleasure.

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[1] See *Uric Acid*, p. 903. The instrument is made by T. Hawksley, 357, Oxford Street, W.

Lastly, these changes in the quantity of uric acid in the blood and the alterations in the rate and completeness of its capillary circulation affect the quality of the blood itself, and thus uric acid is the cause of all the common forms of anæmia. Thus the "blood-decimal" which is obtained by dividing the percentage hæmoglobin by the percentage of cells is found not only to vary up and down from morning to evening with the above factors, but by means of drugs on the one hand, or by giving uric acid on the other, it also can be made almost anything we like, as is the case with the capillary reflux, the blood-pressure, the granules, and urinary water mentioned above. And this has led me to produce a card[1] for the approximate estimation of the blood-decimal from the colour of the tongue, gums, &c., because of the valuable information we thus obtain as to the quantity of uric acid that is or has been passing through the blood.

Thus, while the capillary reflux will tell us the quantity of uric acid in the blood at any given moment, and enable us at once to distinguish between Bright's disease on the one hand and fever on the other, the blood-decimal will tell us whether much uric acid has been passing through the blood in hours, days, or weeks, according to the intervals at which we examine it; and I commonly make use of the card to tell me from the colour-records it gives whether patients whom I only see after intervals of weeks or months are doing well, or otherwise; *i.e.*, whether their blood-decimal is going steadily up—the general result of stopping the introduction of uric acid—or whether it is stationary or descending, owing to their having perhaps a very large store of uric acid in their bodies which is constantly passing through the blood.

We have seen, then, as regards causation :—

(1) That gout is due to poisoning by flesh and tea and

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[1] See *Uric Acid*, p. 573. The card may be obtained from Messrs. Bale and Danielsson, 85, Great Titchfield Street, W.

similar substances, which introduce uric acid into the body in very considerable quantities;

(2) That the uric acid so introduced may not only remain in the body, but may prevent the excretion of the uric acid formed in the body;

(3) That as a result the tissues of the body become more or less saturated with uric acid, which may irritate its fibrous tissues (gout or rheumatism), or may obstruct its capillaries, causing high blood-pressure and defective capillary circulation and their results, such as the great group of circulation-diseases, the uric-acid headache, epilepsy and mental disease, anæmia, Bright's disease, Raynaud's disease, &c.; all of them being mere results of the enormous influence which uric acid exerts on the circulation of the body by obstructing its capillaries;

(4) That all these troubles which have been called by so many names, fall into two groups: (1), the local group; and (2), the circulation-group; of which, the latter is by far the more extensive and important; so that what we used to call gout and rheumatism become almost insignificant by comparison. In the future they will all, I hope, be called uric-acid poisoning, and be recognised as due to poisonous or unnatural food.

The proof of this lies in our new possibilities of prevention and cure, for real knowledge of the real truth always means power; when there is no power there is no real knowledge.

Arthritis due to uric acid is affected by solvents and by precipitants. Arthritis with conditions of normal nutrition is made worse by precipitants, better by solvents. Arthritis with debility is made better by precipitants and worse by some, if not all, solvents. Arthritis which is not affected by either solvents or precipitants is not due to uric acid. But the troubles which we used to call gout, rheumatism, and rheumatoid arthritis are all so affected and are due to uric acid along with varying conditions of age and nutrition, some of which I have mentioned in previous pages.

Some people, I believe, are still in doubt as to the toxicity of uric acid. I am certain that they would not be in that position if they had had a tithe of my own experiences. These experiences have amply proved to me that uric acid in the blood is the cause of many deadly troubles, and uric acid in the fibrous tissues is the invariable cause of more or less serious local irritation and pain. But anyone who is really anxious to understand these matters can quite easily demonstrate it for himself and on himself in the course of a few hours or days. Indeed, it is only possible for anyone to remain sceptical as to the toxicity of uric acid by refusing to look at the effects which are now quite evident all around him and in all directions. But supposing that such a person exists, I would advise him to set about obtaining that convincing demonstration in the following way. Let him take first of all his ordinary capillary refluxes for two or three days, morning and evening, and put them down in curves similar to those that I have given in the figures of my book. After this let him take for another three days twenty grains of bicarbonate of soda three times a day, one hour before each meal, and let him again observe the effects of this alkali on the capillary circulation. When this has been continued for three days, and while still keeping on with the alkali, let him begin to swallow three times a day with or after each meal some three or four grains of uric acid, and let him continue this also for two or three days, again watching the effects on the circulation, on the capillary reflux, and, if he is able to, on the blood-decimal. I think there is no doubt that he will find that this greatly influences his circulation, and that it has important influence on his blood-pressure; and if he has a record of his blood-condition before he began these experiences he will find that this also is getting steadily and considerably worse day by day as he continues to swallow the uric acid. Then let him, still taking the uric acid, give up the alkali and take in its place before each meal, some ten to fifteen drops of nitro-hydrochloric acid, and if he has ever had any pain in his fibrous tissues, any lumbago, any arthritis, I am perfectly certain that he will not have long to wait for a repetition of these experiences and their steady and decided increase. Or in place of the dilute acid he may take if he pleases a small dose of salicylate of soda, say fifteen grains three times a day after meals; and if he takes this dose immediately on leaving off the bicarbonate of soda he will then also for a few hours have a very decided reminder, either in his joints or in his other fibrous tissues, of the local irritation which uric acid produces. After such a trial those who are still sceptical can if they please continue the alkali and take a much larger dose of uric acid than that mentioned—but they will not do this by my advice, because I regard it as a distinctly dangerous experiment—and they may then be convinced of the toxic effects of uric acid in ways that will be both unpleasant and serious for themselves, *e.g.*, syncope, fits, or sudden and severe coma.[1] But in my opinion it is never necessary to demonstrate the toxicity of uric acid by these destructive experiments. It is quite sufficiently demonstrated in the numerous "diseases" from which we suffer to-day as the result of swallowing it. A far more useful and beautiful experiment, though it takes somewhat longer to perform, is to shut out all unnecessary uric acid from the food and to watch day by day, and week by week, and month by month, how all these troubles of circulation, of nutrition, of blood-colour, and of all forms of arthritis and fibrous-tissue irritation, gradually clear up and disappear as the patient becomes free from uric acid and its lifelong poisonous effects. Those who have never seen human blood which is free from uric acid and has been so for years do not realise that the blood of other people may be so much better than their own, nor do they understand what it is to be absolutely free from arthritis, fatigue, stiffness, mental depression and anæmia.

When you can swallow a dose of any substance and can see it day by day slowing your circulation and bringing down the value of your blood-decimal, it is surely waste of time to argue that the substance is not a poison; and the fact that it has been habitually swallowed for many years with results which are increasingly disastrous affords but a further demonstration of toxicity. The demonstration is absolutely complete; those only do not see it who do not look at it: and the uric acid so taken can be watched from hour to hour as it passes through the blood and slows the capillary circulation.[2]

PROPHYLAXIS.

Prevention follows so simply from the above considerations, that it need not detain us. It consists in removing from the diet of the young all substances which contain a notable quantity of uric acid. This is, I believe, not only invariably successful in warding off the above forms of poisoning, but it conduces through its effects on the circulation to a glorious development, both of mind and body, to which in the past we have been strangers. And when the change has been made and become general, future generations will look back on our present records of sorrow, suffering, and degradation with amazement.

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[1] See *Uric Acid*, pp. 386 *seq.* and 714.

[2] See *Uric Acid*, Fig. 75.

TREATMENT.

Treatment again, follows simply from causation, and we have only two things to do:—

(1) To cut off the poisons that have been the cause of the trouble, so that introduction shall as far as possible cease; and

(2) To provide for the elimination of the poison already in the body.

Diet meets the first indication, and for its alterations and the methods of carrying them out I must refer to my book on *Diet and Food.* It is merely necessary to see that a proper quantity of albumens is taken in relation to the weight, and that these albumens are got from substances that contain little or no uric acid, which are briefly bread-stuffs (all forms of white bread, biscuits, and puddings), milk, cheese, dried fruits, and nuts, with a little fresh vegetable and fresh fruit as sauces to them.

One result of thus altering the diet will be that the body will gradually clear itself of the accumulated uric acid which it contains, for uric acid introduced interferes, as we have seen, with the elimination and excretion of uric acid formed or contained in the body. It follows that all diseases due to excess of uric acid in the tissues or blood will slowly get better as the result of altering diet alone. And this we find to be the case. Joints slowly get less stiff and painful, recurrent attacks of irritation gradually become less painful and prolonged, till at the end of 18 to 24 months such manifestations often cease. On the side of the circulation similar slow changes ensue, the capillary reflux gradually quickens, the blood-pressure falls, the colour improves, and the blood-decimal rises in value; in some cases, even in 6 months, the capillary reflux and blood-pressure may have moved more than half-way to normal, and by the end of 12 to 18 months the blood-decimal ought to have gone up at least 20 per cent. I say "ought," for this depends in part on nutrition, and if the albumens are deficient the decimal may remain stationary or rise but little; hence by my "colour card" I can gauge, not only the quantity of uric acid, but the sufficiency of the food.

It is only, therefore, in a few special cases that we need to use drugs at all, for in the majority nature will put things right if you free her from the poisons by ceasing to swallow them.

Acute arthritis is one of these conditions, and the drug (a salicylate) that is used in the acute arthritis called rheumatism is in my opinion the best drug for all arthritis. The only points that require attention are to give the salicylates alone (or if with other drugs not on any account with alkalies, which interfere with their action); and also to give enough of them. One of the best proofs that the arthritis of acute rheumatism is due to uric acid is the fact that, while the arthritis is at once subdued by a salicylate in proper doses and alone, it may not be subdued at all, but may be even made worse if the salicylates are given with alkalies, or if the patients are kept hot and allowed to perspire, conditions which increase the alkalinity of the blood. Hence also salicylates are of but little use in the acute arthritis seen in hot climates, or in this country in very hot weather, unless special precautions are taken to keep the patient cool. If, however, rheumatic fever were due to a microbe, as some suppose, and if the salicylates acted as antiseptics merely, then they should produce as good effects in hot climates as in cold, and with alkalies as without. Clinical experience, however, does not show this to be the case, and the explanation as regards uric acid is simple enough, for salicylates with alkalies, or in conditions of heat and perspiration, are not solvents of uric acid, and do not increase its excretion in the urine; but given with acids or in cold weather, which diminishes the alkalinity of the blood, or in fever with low alkalinity of the blood, they are good solvents of uric acid and greatly increase its excretion in the urine.

My old law, formulated some years ago, was "All substances which increase the solubility of uric acid in the blood do good in the arthritis which is due to it," and under some conditions salicylates are not solvents, and, therefore, do harm, not good. Alkalies are solvents by themselves, and salicylates by themselves; but the two together are not solvents, do not increase the solubility and excretion of uric acid, and, therefore, do harm, not good. Everyone knows that salicylates act much better and with greater certainty in acute arthritis with high temperature than in chronic arthritis with normal or subnormal temperature; and the explanation is again simple, for fever means quickened metabolism with its result, increased acidity of the urine and diminished alkalinity of the blood, conditions which favour the solvent action of salicylates.

In subacute and chronic conditions we have to do all we can to diminish the alkalinity of the blood, which is high, in order to favour the best action of salicylates. Exactly the same holds for the acute arthritis called gout; give salicylates alone and plenty of them, and on no account with alkali or colchicum, as these increase the alkalinity of the blood.

Again, do not, when giving salicylates, cut off wine and cut down meat; my gouty patients are told to keep on with both as usual, for both tend to keep up the acidity of the urine and diminish the alkalinity of the blood, which is what we want with salicylates. The time to alter to a uric-acid-free diet is later on when the salicylates have cleared out most of the uric acid, and can be left off as the arthritis has gone.

If for the same reason I am called to see a case of gout that has already had colchicum, I feel that I see it too late to give salicylates; the field has been spoilt for them. Possibly, by giving acids and wine, and, perhaps, some opium with the salicylates to counteract the evil effects of the colchicum, they may still succeed; but this cannot be counted on with certainty. If the colchicum has obviously produced much depression, and if for any reason the wine, opium, &c., cannot be given, it is better, in my experience, to continue the colchicum and alkalies, and not to attempt to give salicylates at all. These latter are far the best drugs if the conditions favour their action, but there are other conditions (debility, heat, perspiration) in which they can only do harm and make the arthritis worse (*see* Cases in *Uric Acid*, p. 845).

What has to be always remembered is that acute conditions with fever and high acidity of urine favour the best action of salicylates: while conditions of debility with normal or sub-normal temperature, perspiration and all that favours perspiration (as hot climates), with slightly acid or alkaline urine, are most unfavourable to their action as solvents. And this holds for all diseases that are due to uric acid and not merely for arthritis; and in all, the reaction of the urine may be taken as a useful guide as to whether it is right to administer or to withhold these substances.

I may sum up, then, in a few words the treatment of all uric-acid arthritis by saying that in acute cases with fever the indication is to give a solvent and that the best solvent is a salicylate, either in the form of salicylate of soda or salicylic acid, or, perhaps better than either, aspirin; and to give this in sufficient quantity, not less than a drachm a day, and often more than this up to the ordinary doses given in acute rheumatism. In chronic arthritis without fever, but without debility, the same drugs may be given, though care must be taken to feed up, to give a certain amount of acid, to keep the patient cool, and to give some tonics. In very chronic arthritis with marked debility and anæmia the first thing to do is to give tonics and to feed up on any food without the least regard to uric acid; and when the patient has been got into a condition of good nutrition by these means salicylates in one of the above forms may be given with advantage. Where there is marked debility with low acidity of the urine, they should certainly not be given at first; and as I have pointed out above many of these cases of arthritis with debility improve very greatly by feeding up and tonics, because these clear the blood of floating uric acid, and thus put a stop to the "uric-acid filter" action which was the cause of the chronic trouble. In these conditions associated with debility drugs that put a stop to collæmia may produce a more or less acute arthritis as they clear up the collæmia. If they do this, and if there is with the arthritis a rise of temperature, then it would be right to treat the arthritis with salicylates, just as in the cases which are acute from the beginning. But it is a very interesting experience to watch the effect of giving, say, a metal which forms an insoluble compound with uric acid and drives it out of the blood into the fibrous tissues or joints producing the whole series of changes above described in the blood and circulation and in the urine, which may be easily observed as they pass before you. The patient will then tell you that he has had a relapse of his gout, and you will know why he has had it, and you will know also exactly what you have got to do to prevent all future similar relapses.

The many errors and misunderstandings that have arisen as to the ætiology and pathology of arthritis and collæmia, or the local group and the circulatory group of uric-acid poisonings have for the most part been due to the original errors and erroneous conclusions of those physiologists and experimenters who worked without any regard to the solubility of uric acid in the blood and tissue-fluids of the body. If these experiments had never been made we should have seen much more quickly, what we know to-day, that these things are not diseases at all, but mere symptoms of food-poisoning, and that the poisons being left out the symptoms cease to appear.

When this has become common knowledge, it will be obvious that the "uric-acid diathesis" is a myth and that gout is but a symptom of poisoning by flesh and tea; while, lastly, this complete and correct knowledge will give us practically complete power over the symptoms, and enable us to remove all results, except the larger structural lesions, which the food-poisons may have produced.

METABOLISM IN GOUT, AND THE NEED FOR COMBINED INVESTIGATION.

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A CONSIDERATION of general metabolic processes suggests, perhaps indirectly, the line of treatment in many cases of gout. In the absence of any known specific cause, the obvious therapeutic indications are to promote absorption, assimilation, and excretion, and thus assist the natural forces in their resistance to the selective toxines and combinations of complex conditions which together comprise the ætiological factors in gout and its allied affections. Thus, whether the case is one of hereditary gout in its earlier stages or of acquired gout in its later manifestations, the best results are attained when dietetic experiences have determined the level of individual nutrition at which the metabolic organs perform their function with the greatest efficiency.

Under normal circumstances a man weighing eleven stone and performing average work requires twenty grams of nitrogen and 300 grams of carbon per day. In order that these quantities may be easily absorbed and assimilated, the food must be mixed, appetising, and taken at regular intervals. Hutchison gives the following menu as expressing the necessary amounts of carbon and nitrogen :—

Breakfast.	Two slices of bread and butter. Two eggs.
Dinner.	One plateful of potato soup. Four ounces of meat with some fat. Four moderate sized potatoes. One slice of bread and butter.
Tea.	A glass of milk and two slices of bread and butter.
Supper.	Two slices of bread and butter. Two ounces of cheese.

The way in which the body would deal with such a diet may be shown as follows :—

—	Income.		Output.		
	—	Calories.	Urine.	Fæces.	Respiration.
Nitrogen -	21 grams	3,100	19·5 gms.	1·5 gms.	—
Carbon -	335 "		9·0 "	6·0 gms.	295 gms.

and the nitrogenous constituents of the urine would consist of

Urea - - - -	35·45 grams.
Ammonia - - -	0·7 "
Kreatinin - - -	1·0 "
Uric acid - - -	0·7 "
Xanthin bases - - -	0·05 "

The nitrogen-intake and output tends to preserve an equilibrium, although slight daily variations in retention or excess-excretion may normally occur. Carbon, on the other hand, if not oxidised for immediate needs, is stored as "fat' or "glycogen."

In simple starvation the *tissue*-proteids are broken down to replace the food-proteids, the urea is diminished, the ammonia is increased (2–5 per cent.), and the uric-acid output is lessened. Gout, however, cannot be regarded as a state of simple malnutrition; it is rather a condition of insufficient cellular resistance against the absorption of intestinal poisons or auto-toxines, characterised by the production of imperfectly formed metabolites, which act upon some tissues as irritants and in others excite degenerative changes that permit uratic infiltration. As the processes of absorption, assimilation, and elimination are closely associated, quantitative changes in any one function will produce qualitative changes in the other two. So while the increased activities of the purely metabolic organs may lead to widespread cellular changes, the results produced, themselves sufficiently abnormal, may be complicated by the secondary factors of deficient ingestion or excretion.

The elimination of carbon is usually determined by the estimation of the carbon dioxide in expired air and the carbon of the urine; that of nitrogen requires the combined investigation of the total nitrogen, the phosphorus and the sulphur (arising from the complex proteid), and also the determination of the uric acid and xanthin bases (viz., the total purins) derived from cell-nucleins.

RESPIRATORY EXCHANGES IN GOUT.

The extent and rapidity of gaseous exchange may be taken as a reliable index of general tissue-oxidation, and hence respiratory alterations arising from the presence of imperfectly metabolised bodies in the lymph or blood-stream have a special significance.

During the acute attacks, Magnus Levy has shown that the oxygen-intake may be increased by 5—10 per cent., but in chronic gout Klemperer, Levy, and others have found only slight variations from the normal. My own experiments on the action of nuclein-derivatives on the elimination of CO_2 showed that large doses of guanin and hypoxanthin could be taken without any effect. A slight increase occurred after the ingestion of uric acid, but this was due to irritation of the gastric mucosa rather than a direct result of the uric acid, for during investigations conducted with a view to the estimation of unabsorbed uric acid in the fæces, there was pronounced rectal irritation at the time when the unabsorbed portion of uric acid should have reached the lower portion of the intestine. Caffeine produced a marked increase of CO_2-excretion, but, as elsewhere shown, this was due to its methyl attachments rather than to its xanthin- or purin-nucleus. Thus in gout there is usually neither diminished nor retarded oxidation.

ABSORPTION IN GOUT.

The fæces of gouty patients frequently contain increased quantities of fat, and the percentage of unabsorbed nitrogen rises from about 1 per cent. to 2·5 per cent.; further, the researches of His, Von Noorden, and Kaufmann and Mohr show that the powers of absorption diminish as the disease progresses. This increase of nitrogen, however, is not due to food-nitrogen alone, for it may arise from augmented quantities of digestive secretions, from excessive epithelial desquamation or as a result of intestinal putrefaction. The actual loss of unabsorbed food is of less importance than the evident cellular deficiency which may permit the passage of extrinsic toxines or imperfectly digested substances into the general tissues. An estimate of the exact extent of intestinal derangement is hence of more value than a determination of the quantity of unabsorbed proteid. The amount of shed epithelium and perhaps of certain intestinal juices and bacterial products may be better appraised by investigations into the purins of the fæces, since these in part represent the nuclein-contents of the desquamated cells. In some recent experiments I have found that in the fæces there is a somewhat constant average daily excretion of 0, 02—0, 04 gram purin- or xanthin-nitrogen upon a mixed diet. The observations were made over long periods, under varied conditions, and with distinct intervals between each experiment. From other results obtained it is manifest that the healthy intestinal epithelium manifests certain selective powers, and that the absorbed food-nucleins depend either upon the individual nuclein-base or upon the constitution of the particular nucleo-proteid. The absorption of nucleins in gout is still under investigation.

PROTEID METABOLISM IN GOUT.

In gout there is no such extensive disturbance of general metabolism as, for instance, occurs in diabetes or pernicious anæmia, although the frequent association of diabetes and gout suggests the possibility of further metabolic changes. There is rather an instability of proteid metabolism which permits sudden and extensive variations, and predisposes to abnormal products and unusual organic combinations. The difficulty of conducting experiments in this regard lies in the fact that the majority of cases of chronic gout are complicated by parenchymatous and interstitial nephritis. Renal changes lead to such pronounced daily variations in nitrogenous excretion that it is practically impossible to ascribe any abnormal elimination to the primary cause of the kidney-lesion. In the acute attacks, however, there is a distinctly increased output of nitrogen even when the food-proteid is diminished; this probably arises from some toxic destruction of cell-protoplasm, for after the attack there is considerable retention of nitrogen. Little evidence of any "acidosis" or acid intoxication is met with, and the retention may be due to a natural reconstitution of nitrogenous equilibrum such as usually occurs during convalescence. Thus it is probable that proteid metabolism in gout differs from the normal rather in the altered affinities of the metabolites than in their quantitative excess or deficiency. When we are able to record observations upon gouty children or upon the uncomplicated periods of acquired gout, this aspect of abnormal metabolism may appear in another light.

THE METABOLISM OF PHOSPHORUS AND SULPHUR IN GOUT.

The phosphorus-equilibrium does not run parallel with that of nitrogen. Phosphorus exists in three forms in the body; as inorganic phosphates, in combination with proteid, and as a constituent of lecithin, nucleoproteid, &c. After absorption it may therefore be immediately excreted as earthy phosphates, or be retained for tissue-requirements. Thus, even with an accurately-estimated intake and output of phosphorus, the balance obtained tells little as to the cause or object of the retention or excessive elimination.

At one time it was considered that the excretion of phosphorus during gouty attacks was normal, but later work has shown with what ease conflicting results may be attained. Hence it appears impossible to ascertain any distinct relation between destruction of nuclein and elimination of phosphorus. From a clinical point of view a considerable amount of information is gained by systematic comparisons of the purin- and phosphate-excretions. By an approximate method in which the total purins and phosphates are estimated at the same time, I have frequently noted excess of phosphates in the urine of gouty patients. Such increases were generally accompanied by a high urea-percentage and marked nervous disturbances, and Dr. Waldstein kindly tells me that he has obtained valuable indications in regard to treatment from daily estimates of phosphates, particularly in cases exhibiting a nervous temperament. The point is worthy of extended inquiry.

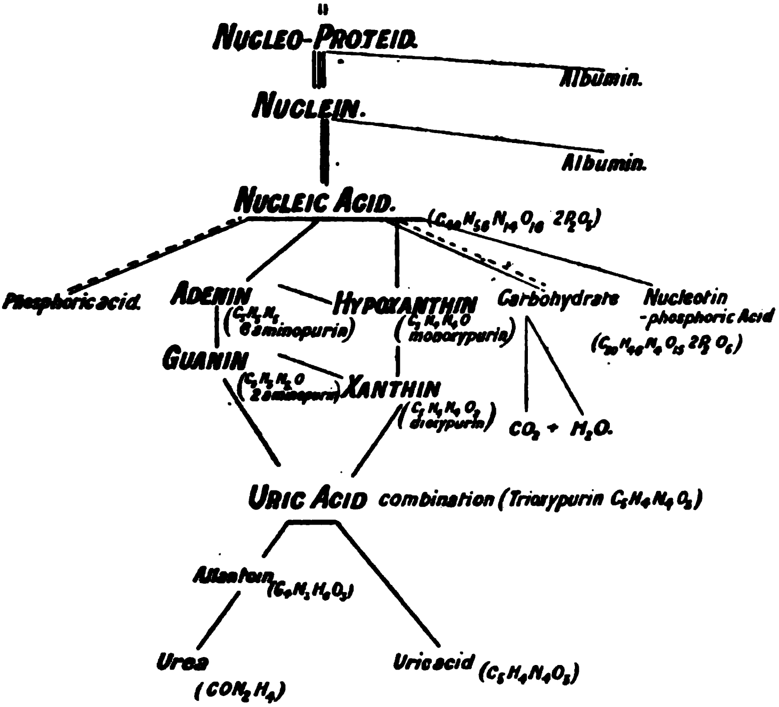
The metabolism of sulphur is chiefly of interest in regard to the aromatic sulphates. These are increased in many gouty attacks and further investigations may provide some definite data for the supposed intestinal origin of the toxæmia present in gout.

NUCLEIN-METABOLISM IN GOUT.

The metabolism of nucleins derives its importance from the fact that it deals with substances yielded by the cleavage of cell-nuclei and the nucleo-proteids of cell-protoplasm. While one may agree with a recent writer who terms the nucleus as a "boarder within a cell," taking its nutrition from the cell-contents and excreting its waste products into the hyaloplasm, it must be remembered that the nucleus is by no means a passive agent. It is actively concerned in the first division of the ovum, in the formation of new cells and in the production of giant-cells; and when degeneration takes place the vital remainder in the nucleus is perhaps the best indication of the possible recovery or death of the matured cell. Thus nuclein-metabolism consists of the synthesis and catabolism of cell-nuclei, and is associated with the life-history of leucocytes, gland-cells, muscle-cells, nerve-cells and the production of red blood-corpuscles. The brief existence and early disintegration of leucocytes is not comparable with the tenure of the fixed tissue-cells, and it is thus conceivable that the daily wear and tear of the latter are only sustained by replacement of constituent atoms. The discovery of intracellular canalisation lends support to such a supposition, for the canaliculi are primarily grouped around the nucleus and most probably perform the function of metabolic channels. Hence the processes of intranuclear catabolism and synthesis are possibly of normal occurrence. That synthesis of nuclein may take place is known from the experiments of Burian and others, the former of whom estimated the average nuclein-yield of the entire bodies of a number of newborn rats from the same litter, and then determined the average amount present in the remaining animals after some weeks' growth. The increase of nuclein-derivatives obtained from the latter was so great (the food in the interim was milk only) that no doubt remained of the possible synthesis of nuclein from simple proteid. Thus in conditions of increased leucocytic destruction and in leuco cythæmia there is an abnormal synthesis as well as disintegration, a need for increased proteid-absorption with a view to nuclein-production, as well as for competent excretion of nitrogenous matter and nuclein, since exogenous nuclein is not used for cell-purposes.

The precise chemical or physical processes which constitute the basis of nuclear action are not known, but it is obvious that if the products of nuclear activity could be quantitatively measured, their determination would lead to a better estimate of cell-efficiency than would the estimations of the urea or total-nitrogen output, for the urea represents proteid metabolism alone, and the total nitrogen includes also kreatinin and other nitrogenous combinations. Naturally, such data would vary when obtained from different individuals, and when pathological processes, local or general, complicated normal conditions, but these variations would soon permit a certain degree of classification and even serve as valuable indications.

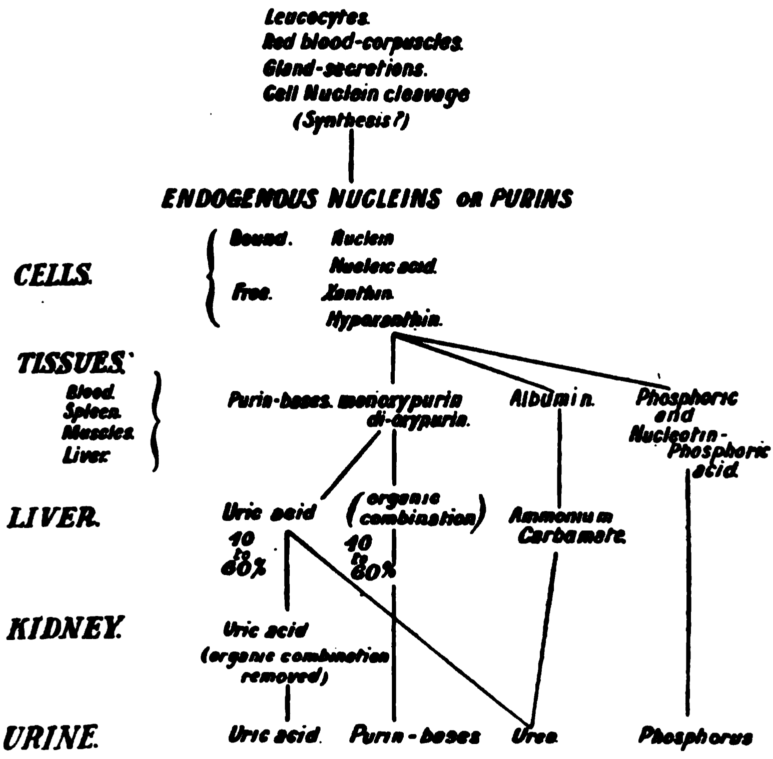
The decompositions which nucleo-proteid probably undergoes in the body may be seen from the following scheme; the organic derivatives of nuclein have been termed "xanthin-bases," the xanthin-bases and uric acid have been together termed alloxur-bodies, and the latter have more recently been called "purin-bodies," because they all contain a nucleus C_5 N_4,


NUCLEO-PROTEID.
Albumin.
NUCLEIN.
Albumin.
NUCLEIC ACID.
($C_{40}H_{56}N_{14}O_{16}$ $2P_2O_5$)
Phosphoric acid.
ADENIN
($C_5H_5N_5$ 6 aminopurin)
HYPOXANTHIN
($C_5H_4N_4O$ monoxypurin)
Carbohydrate
Nucleotin -phosphoric Acid
($C_{30}H_{46}N_4O_{15}$ $2P_2O_5$)
GUANIN
($C_5H_5N_5O$ 2 aminopurin)
XANTHIN
($C_5H_4N_4O_2$ dioxypurin)
CO_2 + H_2O.
URIC ACID combination (Trioxypurin $C_5H_4N_4O_3$)
Allantoin ($C_4N_4H_6O_3$)
Urea
(CON_2H_4)
Uric acid ($C_5H_4N_4O_3$)

to which Fischer has given the name purin. The attachment of side-chains to the central ring and the substitution of other groups explain the production of oxypurins and aminopurins.

By the use of a diet free from purin-bodies it has been found possible to estimate the quantity of nuclein-derivatives or purin-bodies which arise solely as a result of cellular processes. Several experiments have shown that while the diet may vary considerably as to its calorific constituents, without affecting the amount of endogenous purin-excretion, similar habits and conditions of work must be observed. Without committal as to the exact details of processes of which only the bald outlines are known, the following table may be taken to represent the origin and fate of the endogenous purins. It has been already shown that the cell-nucleins are synthesised in the body, and it is probable

ORIGIN AND FATE OF ENDOGENOUS PURINS OR NUCLEINS.



that the compounds pyrimidin, cytosin and uracil, isolated by Kossel, Steudel, and Ascoli from nuclein-rich tissues, are produced as intermediate substances between tissue-proteid and nucleo-proteid. If such is correct, then another set of possibilities occurs for the formation of abnormal or imperfect metabolites. The close chemical relation between uracil and uric acid also suggests a further source of uric acid in metabolic disorders; in such case the uric acid would be formed without transmission through the nuclein-stage. Of course this is pure speculation, but it is based upon evidence which is absolutely material, and which thus inspires more hope of demonstrating the actual cause of gout than the theories previously advanced.

The endogenous purins vary from 0·10 gram N. to 0·20 gram N. during the twenty-four hours. This is evident from the following observations:—

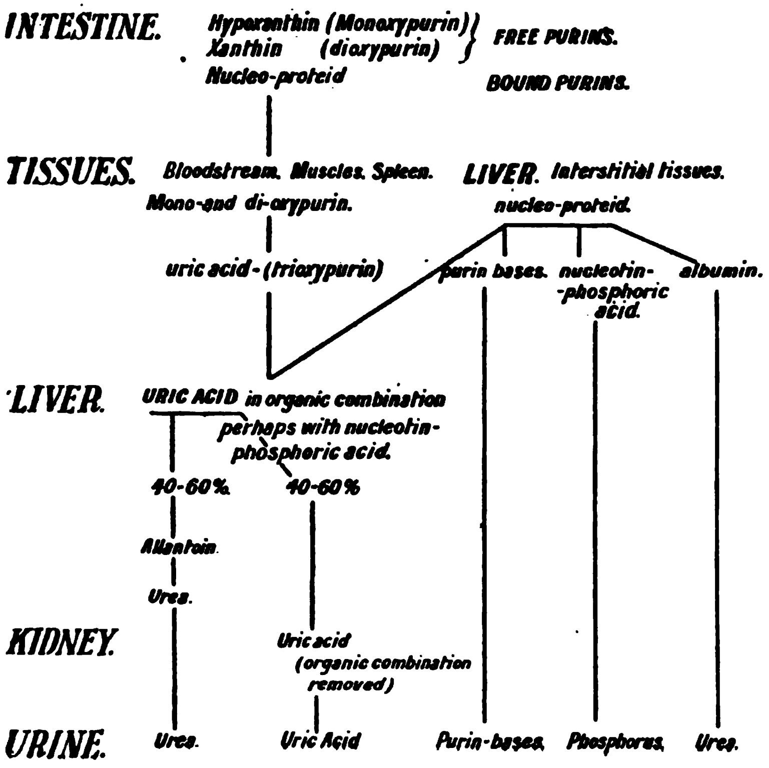
Age.	Endogenous Urinary Purin.	Observer.
Infant, 12 months -	0·0350 gram purin-N.	Camerer.
Female, 7 years -	0·1200 ,, ,, ,,	Walker Hall.
,, 32 ,, -	0·1450 ,, ,, ,,	Walker Hall.
Male 23 ,, -	0·1530 ,, ,, ,,	Burian.
,, 32 ,, -	0·1625 ,, ,, ,,	Walker Hall.
,, 28 ,, -	0·2020 ,, ,, ,,	Burian.
,, 52 ,, -	0·2010 ,, ,, ,,	Kaufmann and Mohr.
,, 72 ,, -	0·1820 ,, ,, ,,	Kaufmann and Mohr.

It is possible for the endogenous purin-excretion to be determined when a known quantity of nuclein is taken with the food, but this is discussed later. The quantity of endogenous purin which appears in the urine only represents a certain percentage of the whole amount of nuclein decomposed in the body; the remainder is further metabolised by the liver-cells and excreted as urea or as bodies intermediate between purin-bases on the one hand, and uric acid and urea on the other. Of the total purin of the urine, nine-tenths are present as uric acid and one-tenth as xanthin- or purin-bases.

In animals a greater percentage of purin-bodies is decomposed into the intermediate body, allantoin, and it is therefore probable that in these the liver effects the cleavage of the uric-acid organic combination more thoroughly than the liver of man.

When man adds animal tissues to his dietary he ingests the endogenous nucleins and purins of another organism; to him, of course, they become exogenous in nature, but as, unlike pure proteid, they are only slightly nutritive, their transformation into combinations suitable for rapid excretion becomes a matter of urgent moment. The probable course followed by the exogenous purin-bodies is shown in the following table:—

EXOGENOUS FOOD-PURINS OR NUCLEINS.


INTESTINE.
Hypoxanthin (Monoxypurin)
Xanthin (dioxypurin)
FREE PURINS.
Nucleo-proteid
BOUND PURINS.
TISSUES.
Bloodstream. Muscles. Spleen.
Mono- and di-oxypurin.
LIVER. Interstitial tissues.
nucleo-proteid.
uric acid - (trioxypurin)
purin bases.
nucleotin- -phosphoric acid.
albumin.
LIVER.
URIC ACID in organic combination
perhaps with nucleotin-phosphoric acid.
40-60%.
40-60%
Allantoin.
Urea.
KIDNEY.
Uric acid
(organic combination removed)
URINE.
Urea.
Uric Acid
Purin-bases.
Phosphorus.
Urea.

The liberation of the purin-bodies from the cells in the case of the endogenous purins, or the absorption from the intestinal tract in that of the exogenous purins, leads to their presence in the lymph and ultimately in the blood-stream. Bainbridge suggests that all metabolic processes are accompanied by increased lymph-flow from the changed osmotic conditions following the production of large quantities of substances of low molecular constitution or crystalloid form; so the purin-bodies may indirectly stimulate lymph-flow. In the tissue-spaces, they are probably to a slight extent oxidised to uric acid, but the liver is without doubt the organ which plays the chief part in the elimination of these waste products. The purin-bodies leave the liver in some form of uric-acid organic combination or as intermediate bodies between uric acid and urea. The uric-acid resultant from the intake of exogenous oxy-purins varies from 40 to 60 per cent. of their total purin-content, as will be seen from the appended table taken from Burian and

Observer.	Exogenous Daily Food-Oxypurin Nitrogen.	Exogenous Urinary-purin Nitrogen.*
Minkowski - -	3·0 gm. Hypoxanthin = 1·23 N.	48·6 per cent.
Burian and Schur -	1·6 gm. ,, = 0·66 N.	46·2 ,,
Krüger and Schmid -	1·5 gm. ,, = 0·62 N.	62·3 ,,
Walker Hall - -	0·5 gm. ,, = 0·20 N.	60·0 ,,
Burian and Schur -	300 gm. Beef = 0·18 N.	51·1 ,,
,, ,, -	300 gm. Veal = 0·18 N.	53·9 ,,
,, ,, -	300 gm. Ham = 0·19 N.	63·2 ,,
Walker Hall - -	450 gm. Chicken = 0·23 N.	52·1 ,,
,, - -	537 gm. Plaice = 0·17 N.	58·2 ,,
,, - -	400 gm. Beef = 0·24 N.	47·0 ,,
Kaufmann and Mohr -	100–300 gm. Veal = 0·06–0·18 N.	48·1 ,,
Burian and Schur -	500 gm. Liver = 0·60 N.	52·6 ,,
,, ,, -	400 gm. Spleen = 0·64 N.	52·9 ,,

* This consists of the excess over the average endogenous excretion. Except for the oxypurin specified, the diet was purin-free.

Schur's most recent publication; in the cat and dog, however, the percentages are considerably lessened, and allantoin is met with in large quantities. Allantoin, a simpler body than uric acid, is due to further decomposition of uric acid; hence nuclein-metabolism is more active in the cat and dog, and perhaps the comparison of such animal and human excreta expresses the limits of sluggish or active metabolism.

The amino-purins, guanin and adenin are not well absorbed and are probably excreted as oxy- or methyl-amino-purins. The nucleo-proteids of thymus and pancreas yield only 25 per cent. of their total nuclein-nitrogen as urinary uric acid.

The presence of these exogenous purins in the blood-stream in the form of uric acid and xanthin-bases is not indicated by the usual tests for these substances, and it is generally understood that uric acid cannot be demonstrated in healthy blood. This is probably due to some loose organic combination with the purin—Minkowski suggests the purin-base, free nucleotin-phosphoric acid—which hinders precipitation by the usual reagents. When, however, after excessive nuclein-ingestion, or after increased nuclein-formation, as in leuchæmia, this organic combination is insufficient to prevent precipitation, the presence of uric acid is easily shown. The blood, however, is rarely a saturated solution of uric acid or purin-bodies, for Klemperer has shown that freshly-drawn blood will always dissolve added uric acid: hence it is not improbable that the deposition of urates in the tissues, if not primarily arising from tissue-necrosis, is a tissue-infiltration dependent upon qualitative affinities rather than quantitative excesses of purins in the blood or lymph.

Normally, the exogenous purins are rapidly removed from the system. Nucleo-proteid, however, requires 24–30 hours. The healthy kidney readily excretes large quantities of purin-bodies. In delayed elimination, except when definite renal changes are present, the liver is more frequently at fault.

The Exogenous Purins in Gout.—Since the definite division of purin-bodies into "*endogenous*" and "*exogenous*," too little time has elapsed for the record of many observations; but from the studies of Reach, Kaufmann, Vogt, and Chalmers Watson, it may be shown that in gout exogenous nuclein is more slowly excreted than in health, and that in some cases there is distinct retention. These points appear in Reach's case. His patient was 60 years old, had a 20-years' gouty history, slight emphysema, bronchitis, and arterial rigidity; the urine contained traces of albumen, but no formed elements. The excreta may be tabulated as follows:—

—	Total Nitrogen.	Uric Acid N.	Expected Excretion.
Fixed diet for 5 days	58·2 gms.	0·233 gms. N.	—
Fixed diet for 8 days plus 150 gms. pancreas per day.	89·1 ,,	0·671 gms. N.	1·124 gms. N
Fixed diet for 6 days	65·3 ,,	0·495 gms. N.	0·294 gms. N.

In this case there was a retention of 0·453 gm. N. during the eight days' experiment, and a surplus excretion of 0·201 gm. N. during the third period, although with this excess the total was still short by 0·253 gm. N.; hence, for the whole period there was retention of 0·253 gm. N. Probably there was more than this, for during the third period an acute attack supervened.

Vogt compared the results of rich purin-holding food upon a gouty patient and a healthy individual at the same time. The gouty patient showed retention, and delayed excretion of purin-bodies. These studies confirm clinical experiences, and there is thus little doubt that the gouty individual does not well metabolise exogenous purins. But the existing experimental results are too few to admit reliable inferences, and only broad deductions can be made by comparing the metabolic activities of different individuals. If these recent scientific advances are to have practical application, a comparison of the results of metabolic experiments upon the same person, at different periods of life and under varied conditions of health, should be made.

We can, however, safely admit that exogenous purins lead to excess of nuclein-derivatives in the blood-stream, and that their presence can be demonstrated by suitable methods. If so, it may then be asked, Do these substances exert any toxic action upon the tissues? Croftan, Kolisch, and others have produced arterial and renal lesions by injecting hypoxanthin into animals, and the writer has confirmed these results and also obtained early parenchymatous changes in the liver by long-continued injections of hypoxanthin; but in man reliable methods have shown that the purin-bases are rarely present in excess in the blood-stream. Adenin is a marked irritant (Minkowski). Uric acid does not lead to any distinct tissue-lesions, but in the writer's case large doses have always caused irritation of mucous membranes; and the recent case reported by Carrière, as well as the earlier reports of Drummond, indicate that the meninges of the cord and brain are somewhat sensitive to the action of uric acid. Of course, in these cases there may have been a distinct "uric-acid tendency." The writer's experiments upon the effect of the purin-bodies showed that they did not lead to any alterations in the general blood-pressure. As to the form in which uric acid circulates in the tissue-fluids much conjecture exists; the quadri-urate may occur in some parts of the blood-stream, but in others the presence of localised collections of carbonates or phosphates would soon change it into the less soluble bi-urate, and so even under normal conditions the uric acid would circulate in several forms. A considerable amount of evidence has lately accumulated, which suggests that the purin-bases, mono- and di-oxypurin alike with the tri-oxypurin, uric acid, exist in the blood-stream in combination with a complex organic body; hence the views as to different forms of circulating inorganic urate-combinations may soon undergo revision.

There is evidently a definite time-period for the excretion of exogenous purins which varies with each individual, and this point merits extended investigation.

The Endogenous Purins in Gout.—To the earlier records of His, Luff, Magnus Levy, Chalmers Watson and Klemperer, Kaufmann and Mohr have recently added some observations which tend to show that in the intervals between the acute attacks the excretions of uric acid and of the xanthin-bases are practically normal in amount, if not in character. These statements may be accepted for the moment, with the proviso that existing data of personal variations within the limits of health are too few, and that the duration of the experiments cited was too short. Estimations of the endogenous purin of the same individual at brief and extended intervals and under various conditions of reaction, rather than estimations from scattered cases, are necessary for safe deductions.

Prior to the acute attacks the uric-acid excretion is diminished for a few days; it is increased upon the first day of the attack and then gradually regains the usual level of the intervals.

During the attack there are excessive quantities of purin-bodies in the blood-stream, and this condition is associated with a slightly lowered alkalinity. There is, however, no distinct "acidosis." The presence of large amounts of purin-bodies in the tissue-fluids produce no ill effects if the kidney is normal; for instance, gouty symptoms do not always accompany rich nuclein-intake, nor appear in leuchæmic patients. The molecular concentration of the blood is generally normal: there is neither distinct leucocytosis, altered numbers of red blood-corpuscles nor change in the hæmoglobin-coefficient. Chalmers Watson draws attention to certain degenerated myelocytes which occurred in his cases. It would be interesting to compare the peripheral and central arteries and veins to see if these resulted from local processes. The presence of granular kidney leads to variations in the output of nitrogen as well as in that of phosphorus and purin, but in many cases normal phosphorus-excretion with purin-retention has been observed.

Thus during gouty metabolism, so far as the few recorded estimations permit any inference, the elimination of endogenous purins is not appreciably altered from the normal, except in the acute attacks. Even when renal changes supervene, the purin-excretion is less affected than the urea-output.

The endogenous purins cannot be regarded as the causative factor in gout, although without doubt they readily and rapidly express the extent of tissue-toxæmia as measured by cell-destruction; and their excretion thus becomes symptomatic. Whatever the primary cause may be, there is no doubt that the body-cells for a long time successfully compensate for the toxæmia; and it is possible that only when the metabolites become markedly abnormal, do the purin-bodies form unusual organic combinations which hinder renal action and favour their deposition in the tissues.

HEREDITARY AND IRREGULAR GOUT.

Amongst the numerous and diverse opinions upon the ætiology of gout all writers ultimately agree as to our ignorance of the exciting cause or causes, and in regard to its course, pathology and treatment, they consider each case as a law unto itself. The majority of the experiments already stated have been made upon individuals suffering from unmistakable or advanced gout. The observations have been carried out by laboratory or hospital workers upon stray cases; and the results, as a rule, are only comparable with those obtained from other patients. Now there are certain distinct groups of symptoms which appear in the children of gouty parents, and at first these are quite apart from any functional or actual renal deficiency. Luff considers that a functional renal derangement precedes distinct tissue-changes, and that if the exciting cause be removed the kidney recovers its healthy functions. What is the exciting cause? The metabolic organs are hereditarily imperfect, and the results of their deficient action are shown in gouty prodromata. The liver may be indicted as the weak factor in the metabolic chain and charged with inefficient filtration of toxines, intrinsic or extrinsic; but what evidence have we to fall back upon? Of all the varieties of experimental data, those from children are surely the most scanty. We know little of the endogenous output of children, and less of their powers to metabolise exogenous purins. Those who have opportunities to observe these conditions in children, especially in those of gouty stock, would contribute very largely to scientific advancement and towards the rational treatment of gout, were they to record systematically the nitrogenous and purin-output under varied conditions. So many questions might be asked on this almost unexplored portion of the subject. We talk of heredity in gout, but how little is known of the general metabolism of gouty children! And yet it is in this class of patient that the earlier metabolic changes should be most evident. Acquired gout does not as a rule present itself for treatment until an acute manifestation occurs, but the children born in the elective gouty age-period of the parents may reflect the earlier cell-changes, in other words, what is termed the purity of the gouty strain may yield some of its causative factors to investigations upon children.

Perhaps here may be suitably discussed the question of the nervous influences of gouty metabolism. Mental conditions often excite acute exacerbations, and gout is frequent amongst the class who work at high brain-pressure. Whether this is due to actual loss of nervous stimulation to the cells or to the withdrawal of trophic influence is not clear, but the association of neuralgia, sciatica, and even cord-lesions presenting morbid changes, with well-known gouty symptoms, suggests the influence of the primary toxæmia on overworked nerve-centres and the secondary partial removal of trophic control over the metabolic organs.

THE NEED FOR COMBINED INVESTIGATION.

The possibility of endogenous purin-determination has done much toward the ultimate elucidation of the cause of gout, but a large number of observations upon healthy individuals, of varied habits and living under different conditions, ought really to precede any estimations of pathological nuclein-metabolism, and such records should extend over long periods. Afterwards, the association of intestinal disturbances with gouty symptoms and with the output of sulphates and purin; the systematic post-mortem investigation of changes in the liver and pancreas; a study of the definite vascular changes preceding acute attacks or accompanying chronic lesions, of the relation between the excretion of urea and phosphates and the nervous conditions associated with gout, and of the cellular changes of blood and marrow might well repay systematic investigation. But these clinical studies are not available for the average laboratory worker. It is here that the practitioner may find material which will repay observation. With the ureameter, the uricometer or the purinometer, under certain conditions, much valuable information may be collected.

If we are to approach the cause of the gouty toxæmia or unravel the tangle of complex forces which contribute to its origin or its maintenance, it is manifest that we must tackle the early symptoms and particularly those associated with heredity. Could not some Association, Society or specially-formed Committee initiate a combined attack on these urgent and pressing questions? Isolated work on a subject of this nature is economic loss. The nervous strain of existence is increasing, and this already-prevalent disease tends to increase *pari passu.* If the exact points of inquiry were formulated, if details of work were furnished to voluntary observers, if clinical material were collected for laboratory investigation, and the collated results edited and published, some definite advance would certainly follow. It is remarkable what the average patient will do for a definite object when the scientific probabilities are made clear to him, and there is perhaps no subject in which such diverse ætiological opinions are held and of which the early changes have been so little elucidated as that of gout and its allied disorders. We do not lack men for the work. There are numbers who would undertake scientific observations if only definite subjects were assigned to them and a little preliminary help provided. Comparative pathology cannot contribute much in regard to this special lesion; human studies are needed. A scheme such as this should later provide the laboratory worker with many opportunities; for the co-operation ought to result in an increased amount of clinical material specially selected for the various phases of exact investigation.

THERAPEUTIC CONSIDERATIONS.

To prolong the resistance to toxæmia and to sustain trophic tissue-control, it is apparent that the gouty patient should be thoroughly well-fed; and by the term "well fed" is meant the ingestion of a sufficient quantity of easily-assimilated food. It is quite unnecessary to restrict gouty individuals to vegetable proteids; in fact, for a person who digests large quantities of vegetable food and who possesses absorptive epithelium proof against the sulphur-derivatives and the constant irritation of the increased bulk of food necessary for the exclusive vegetarian, gout can have no terrors. Many cases of gout exhibit their worst acute attacks after prolonged periods of vegetable diet. Still, the sufferer from hereditary tendencies or acquired habits must only ingest the exact amount of meat-proteid that he can easily and thoroughly metabolise. Rich, indigestible food will decrease the selective powers of the intestinal cells and increase the work of the liver; too much plain meat will load the tissues with imperfectly-assimilated bodies, flood the blood-stream with abnormally-bound purins, and favour the occurrence of the particular individual symptoms of "uricacidæmia" or predispose to infiltration of cartilage. The line of safety lies in a precise knowledge of the varieties and quantities of meat-foods that each patient effectively metabolises. The maintenance of intestinal secretions and excretions is of first importance; the second point is to determine the relation of elimination to ingestion. Retention of nitrogen and particularly of purin-nitrogen is to be avoided; delay in excretion is one of the early signs of deficient metabolism. Thus it becomes necessary to estimate the endogenous purin-output of each patient, and to observe the time required for the excretion of exogenous purins. For the former purpose opportunities will frequently occur when the patient is on a purin-free diet (milk, eggs, cheese, bread, potatoes, rice, &c.) during convalescence from acute attacks; for the latter, known quantities of purin may be employed, say, for instance, half a gram of hypoxanthin or weighed amounts of meats whose purin-contents may be calculated from available tables,[1] and a comparison made with the endogenous quotient. It is well to measure the twenty-four hours' urine from 8 a.m. to 8 a.m. and make observations upon the total daily urine. The method may appear more clear from the following estimations (*see* p. 80):—

These details may look cumbrous and difficult, but they are not so in actual use. A few personal observations will suffice to convince the most sceptical that much knowledge of vital activities may so be gained and the patient's life be thus regulated on scientific lines without the entire prohibition of natural tastes and inclinations.

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[1] Purin-contents of foods:—
  Purin-free:
    Milk, eggs, butter, cheese, white bread, rice, sago, fruits.
  Under 0·02 per cent. purin N.:
    Beer, stout, onions, asparagus, malted lentils, brown bread.
  Under 0·03 per cent. purin N.:
    Oatmeal, lentils, beans, peas.
  Under 0·05 per cent. purin N.:
    Plaice, salmon, cod, pike, halibut, mutton, veal, pork, ham, turkey, chicken.
  Under 0·1 per cent.:
    Liver, steak, soups.
  Under 0·4 per cent.:
    Sweetbreads.

Diet.	Total Daily Purin-Excretion.	Amount of Exogenous Moiety.	Remarks.
Purin-free	0·1612 gm. N.		Nuclein-metabolism rapid and normal.
,,	0·1625 ,,		
,,	0·1630 ,,		
0·0275 grm. oxypurin	0·3579 ,,	60 per cent.	
Fixed diet	0·1380 gm. N.		Nuclein-metabolism delayed.
,,	0·1560 ,,		
,,	0·1600 ,,		
0·0190 grm oxypurin	0·2010 ,,	26 per cent.	
Fixed diet	0·1900 ,,	21 ,,	
,,	0·1700 ,,	10 ,,	
Fixed diet	0·2010 gm. N.		Nuclein-retention.
0·200 grm. oxypurin	0·1800 ,,		
,, ,,	0·2500 ,,	25 per cent.	
,, ,,	0·1900 ,,	—	
,, ,,	0·2000 ,,		
,, ,,	0·1700 ,,		

May 11th.				May 12th.			
Diet.		Urine.	Total Purins.	Diet.		Urine.	Total Purins.
Fixed.	10.30 a.m.	0·4060 Hypoxanthin = ·1609 N. in 600 cc. water.		Fixed.	10.3 a.m.	600 cc.	Water only.
	11.40 a.m	390 cc.	0·0464 gm. N.		12 noon	315 cc.	0·0590 gm. N.
	12.40 p.m.	230 cc.	0·0454 ,, ,,		1 p.m.	360 cc.	0·0254 ,, ,,
	2.30 p.m.	135 cc.	0·0348 ,, ,,		2 30 p.m.	135 cc.	0·0137 ,, ,,
	5 p.m.	110 cc.	0·0494 ,, ,,		4.30 p.m.	150 cc.	0·0300 ,, ,,
		865 cc.	0·1760 gm. N.			950 cc.	0·1281 gm. N.

It is, perhaps, needless to add that comparative estimations of urea and phosphate will contribute to the indications for specific treatment, and that repeated purin-determinations in each case are advocated in preference to spasmodic observations spread over a number of cases. Should the exogenous or endogenous purins be delayed or retained, their dilution in the tissues by imbibition of large quantities of water or harmless fluids obviously offers the best assistance to the renal cells for effective excretion. The endogenous purins are not directly influenced by diet. Indirectly, they normally depend upon a competent metabolism, and so upon a sufficient supply of assimilable food. In our present state of knowledge their estimations should suggest the extent of tissue-destruction and indicate when intestinal antiseptics should be employed.

NOTES RESPECTING THE DIETARY FOR GOUTILY-DISPOSED PERSONS.

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> "*Some physicians are so regular in proceeding according to art for the disease, as they respect not sufficiently the condition of the patient.*"
>
> Bacon's Essays: "*Of Regimen of Health.*" 1597.

THE fact that gout has long been regarded as a disorder dependent on errors of digestion and systemic metabolism has led to many observations respecting the influence of various articles of diet upon the production and course of the disease. Such a study is no easy matter, and to secure a sound and practical knowledge of it requires long experience, calm judgment, and a mind free from any bias that may tend to pervert it.

It can hardly be doubted that many, if not all, of the dietetic methods practised to-day are founded on the commonly-accepted theory which is held as to the pathogeny of gout,—to wit, its dependence on an abnormal production or disposal in the system of uric acid and its immediate congeners.

In this brief essay, I am not concerned to touch particularly on this point. I have elsewhere expressed my belief that gout is not a purely humoral disorder, but is dependent on a neuro-humoral pathogeny.[1] By most authorities uric acid is regarded as the touchstone of the disorder, and some even go so far as to declare that to establish the diagnosis of gout or goutiness in any instance, there must be demonstrable evidence of this substance. I will only remark in passing that such pedants will have long to wait if they insist on this evidence.

In any case, it is certain, first, that the peccant matter of gout is produced within the body, and is thus auto-toxæmic,

[1] *Trans. XIII. Internat. Med. Congress, Paris,* 1900 &c.

not entering infectively from without as is the case in rheumatic or other varieties of toxæmia ; and, secondly, that the subject of gout, either by inheritance or acquirement, is so far peculiar in his constitution that he reacts differently to various agencies, such as climate, food, &c., from persons not so disposed. The metabolic reactions of the gouty are abnormal.

I am of opinion that many of the methods in practice relating to diet as well as to therapeutics for gout are founded mainly on the principle of treatment for the *disease,* and not for the *patient.* To state this in another way, the *patient* is not regarded in the proper light, or is disregarded, and all the efforts to set him right are directed against the perturbations of uric acid. This is not the true clinical method, and it constitutes a radical fault in practice. Men and women are not so many ninepins or human units invaded by morbid agencies. No two individuals, indeed, are precisely alike in respect of their constitution or metabolic processes. In this sense, then, there is no treatment for gout, and no special diet for the gouty, as such, but there is a treatment and a management for the particular patient who manifests symptoms of gout. The same principle, of course, applies to patients suffering from pneumonia, or from renal, tubercular, or any other maladies. The neglect of the personal factor in each patient narrows the field of observation, and tends to stereotype the practice of those who work in special departments of medicine. The great whole is lost in a part, often a very small part, of the case, and the acumen of a widely-exercised physician is not brought to bear upon it

A study for a few minutes of the peculiarities of gouty patients can hardly justify any dogmatic assertions as to the diet required, or fairly authorise the over-elaborate prescriptions sometimes to be met with, which have been placed in their hands. In many instances these patients have already discovered for themselves what manner of diet best agrees with them. They can often blame no indiscretion or irregularity as to food for the onset of acute, or the continuance of lingering symptoms. Sometimes this personal experience has led them rightly. More often, the articles of food omitted or introduced, have been denounced or recommended by lay friends, or the practice has been gathered from some unauthorised or untrustworthy source, and no benefit has been derived from it.

The histories afforded in some cases reveal at times an extraordinary and even a ludicrous series of recommendations as to diet.

Thus, it is not seldom declared that no red meats are to be taken by gouty persons, but the white meats, fish, and game are allowable. Again, beef is generally condemned, while mutton is regarded as permissible. Some authorities are of opinion that all forms of animal food are gout-provoking. In Germany some physicians condemn fowl as directly productive of the disorder. We may note that all salted food, sweetbread, and other viscera are excluded. With respect to other articles of diet, we find ordinary bread sometimes forbidden, while sugar is almost universally struck off the list of permissible foods. Common salt is excluded, and tea and coffee are regarded by some as direct doses of uric acid. Potatoes are condemned in common with all things that grow underground. Many patients are warned never to eat any kind of fruit. Wine and all fermented liquors are forbidden.

In this strange enumeration I have not drawn upon my imagination, but have simply told of prescriptions from various sources which have come under my own observation. The sad fact remains that, having undergone most of these varied dietetic experiments, the majority of sufferers still remain more or less gouty, and continue to seek further advice. We may well ask how much ordinary food fit for human beings is left for such patients to consume, and wonder whether gouty patients are to be regarded as beings from some other planet, since they can hardly be expected to fight the battle of life on such conditions amidst our present environment.

As I have already stated, these curious prescriptions are the result of efforts to treat gout without reference to the patient. It is sometimes forgotten that in all diseases the best possible nutrition of the body has to be secured. "The King's Government must be carried on." Patients may submit for a time to certain severe deprivations, but seldom for an indefinite period unless they find real benefit from the specific regimen. They not seldom learn for themselves what articles of food disagree with them or tend to provoke gouty symptoms.

We may now inquire more particularly as to the real inwardness of some of the dietetic methods just described.

With respect to *animal food* generally, it may be declared at once that it is not only harmless, but beneficial, to gouty persons, provided it be taken in moderation. The alleged difference between red and white meats I regard as nonsense. Anybody can readily understand that tender mutton is more digestible than tough beef. The notion as to the permissibility of white meats has come in all probability from the Continent, where the ordinary white meat is veal, a very different and superior article of food to any that can be procured in this country, and where beef and mutton are very inferior in texture and flavour to the products of Great Britain and her Colonies. I recognise no difference between red and white meats, fowl, and any variety of game in respect of gout-provoking qualities, provided the quantity consumed is restricted and the cooking is plain and appropriate. Pickled or salted meats are, as a rule, inadvisable, but they need not be absolutely excluded from the dietary. Fish, especially white and fresh, is one of the best articles of diet for the gouty. Lobsters, oysters, and most shell-fish are quite innocuous provided they are eaten in good condition. No viscera need be excluded from the diet.

With regard to *vegetable* food, ordinary bread, plain or toasted, or plain biscuits are, of course, permissible. It is hard to understand why potatoes should have been forbidden. Certainly, the largest eaters of them know nothing of gout. Plainly cooked, they are quite harmless. It may be remarked here that certain dyspeptics are better able to digest separately a meal of proteids or of carbo-hydrate food, and suffer from the effects of a mixture of both, but this disability is not peculiar to gouty subjects. The assertion that roots and vegetables grown underground are harmful I fail to understand, and I have no knowledge of any ill-effects from their employment by the gouty. Fruit is condemned for very inadequate reasons. Some gouty patients are certainly better without any variety of it, but most of them can take fruit, cooked or raw, in moderation at proper times of the day, and apart from meals, not only with impunity, but with benefit. Tea, cocoa, and coffee, properly prepared, are quite harmless : the greatest consumers of these know nothing of gout. Sugar taken in moderation I regard as not only harmless, but beneficial. On theoretical grounds a purely vegetable diet is extolled by some as a means of averting gout. As such a diet, were it attainable, is condemned by physiology, no less than by common sense, for the majority of mankind, it needs no consideration here. Certain vegetable foods are useful for gouty subjects, especially spinach, the various cruciferæ (including the cresses); also celery (preferably cooked). Asparagus should be taken sparingly.

Respecting *wines* and alcoholic liquors generally there is much to be declared. It is certain that the least excess in any of them or the use of inferior wines is harmful. So-called moderate drinkers of wine, as a rule, drink far too much. Many gouty persons are best without any. But the fact remains that many others are the better for the use of a little good wine, taken with one meal in the day. What that wine shall be is a matter of personal experience. The quantity must be well below what is commonly regarded (by men) as moderate. The capacity for adequate and beneficial disposal of it varies much in different individuals, and, further, must vary according to the habits, pursuits, and many personal factors in each case under consideration. There can, therefore, be no universal rule as to kind or amount. Much heed has to be paid to quality, season of the year, and preferences of the patient. Most varieties of malt-liquors are harmful to the majority of gouty subjects, but in summer many can take small quantities of the lighter varieties, such as lager beer, with impunity and benefit. One of the earliest symptoms of the onset of a gouty habit is an inability to digest even small quantities of malt-liquor. Well prepared cyder is an excellent drink in warm weather for many gouty patients, but the quantity should not exceed a pint.

Thus far I have in several respects combated the views which are commonly held regarding the appropriate diet for gouty persons. The main point which stands out is this, that a very large choice remains, which includes most of the edible and potable articles consumed by healthy individuals. What remains to be done is to find the particular diet which is suitable for each individual patient.

The general line of treatment in all cases is based on an effort to raise the level of health by whatever means are available. When this level is lowered from any cause, gout, or indications of its approach, may be expected to supervene in persons so predisposed. We find in practice that many sufferers are careful and prudent in diet, and commit no indiscretions. Their general tone is low. An improved and adjusted diet is helpful in such cases. To curtail their food and to send them to various spas is not helpful, but generally harmful. The class of gouty persons who live sumptuously, eating what amounts practically to two dinners daily, and habitually consuming what they regard as moderate quantities of light wine or whiskey, require other treatment. This class, in the progress of what we are pleased to call modern civilisation, is a growing one, and the dietetic habits alluded to are not seldom associated with nervous exhaustion induced by strain of business or affairs, by endless rounds of pleasure-seeking, or the engagements of society, and with a deficient amount of sleep.

In these cases we have to moderate the intake of all varieties of food and stimulants, and especially to curtail the vicious items of the fashionable *menu.* Some of the startling prescriptions I have criticised may appeal to a few of these self-indulgent persons, but many of them have already earned strong proclivity to renewed attacks of gout by damage to the integrity and perfection of their hepatic and renal metabolism. They find relief in regular visits to spas which stave off the evil days to come. The mischief commonly manifests itself late in the third decade, and a general plethora, with high arterial tension and a progressive obesity, paves the way to gouty outbursts either overt or larval.

Relief will come readily, not so much by omitting this or that particular article of food, as by a daily and systematic reduction of the whole amount consumed. What has especially to be forbidden is rich, seasoned, greasy, and twice-cooked food. Strong soups, cooked tomatoes, rhubarb, food cooked with fat or sugar, large mixed meals of animal and farinaceous foods, together with fruit and wine—all these are gout-provoking.

As a rule, the meals should consist of fresh food, plainly cooked, as by roasting or grilling. Variety is apt to lead to excess.

Lemon-juice, sometimes freely taken, for pains which are regarded as rheumatic, and are most often gouty, is sometimes very harmful and productive of gouty outbursts. A little good wine is certainly helpful for many elderly patients, but two to six ounces is sufficient, best taken with one meal only in the day. For most patients the wines of Burgundy, the Midi districts, Algeria, Hungary, California, and Australia are unsuitable. Claret, as grown in the Bordeaux district only, may disagree in some cases, but more often, as I believe, because half a bottle of it is taken when two glasses would suffice and be well disposed of. Taken with a little water, it is one of the safest wines to employ, and the same may be said of the lighter wines of the Moselle. There is little if any difference to be noted in the employment of the red or white varieties of wines so far as concerns gouty persons. The best qualities of champagne, ten years old and not too dry, suit many gouty patients very well, but the rule as to quantity must be rigidly observed. No mixing of wines at any meal is permissible. A little mature port wine is often well borne by gouty patients. I know of no special virtue in what is called tawny port wine, sometimes vaunted as "safe" for such persons. Many gouty patients are, however, quite intolerant of the smallest quantities of most of the wines just mentioned. We find, then, no rule in respect of the prescription of wine for the gouty. There can be no such rule. Sir Thomas Watson's advice to any young person showing signs of gout was excellent. He urged such to abstain from wine altogether.

It is to be noted that water-drinkers are apt to be large eaters, and they therefore undo the good effects of their abstention from wine. They generally drink too much water with their meals. Extra water is certainly advisable in most cases, but is best taken early or late in the day or between meals. Hard waters, impregnated with lime, or containing an undue amount of iron, are harmful for persons predisposed to gout. Many gouty patients take lithia-, potass-, or Vichy water at their meals. I believe all of these medicaments are harmful if regarded as articles of diet. It is not becoming to take medicines at meal-times. These waters are apt to prove depressing, and should only be employed temporarily as prescribed.

I suppose the great majority of British gouty patients take, or are urged to take, whiskey in place of all other forms of alcoholic beverages. There is no reason why brandy and gin should not also be used. The difficulty is to secure a pure grape-spirit at a reasonable price. The quantity in any case must be small, and should never exceed two or three ounces, well diluted, and taken only with one meal in the day.

The question of appropriate diet for the gouty patient is therefore a matter of as careful consideration as is the prescription of any particular treatment by drugs. The patient before all, his family history, his up-bringing, his habit of body, his environment, his daily habits, his age, his textural qualities and condition—all these points, personal and individual, have to be regarded and pondered in laying down rules (which in the first instance must always be experimental) for diet. So when we are told that this or that is good or bad for gout, we know that our informer is ignorant of the very alphabet of the matter. We have, in truth, many articles to select from, but the keynote of our prescription should be strict moderation in all or any one of them. I subjoin some remarks made by men very competent to judge, which will sum up for me the substance of the views I have here endeavoured to express. "No second helpings" (Prof. Humphry). "Beware of luncheons; they spoil the frock-coat figure!" (Sir John Banks). "Give your gouty patients a little wine" (Dr. H. G. Sutton). "I am so far from desiring you to abstain from animal food, that I absolutely wish you to eat it freely every day. I am not afraid of your indulging the cravings of your appetite, provided that it be not stimulated by heating condiments or strong liquors, and that it be not excited by that elegant but useless variety, with which modern luxury has covered the tables of the rich. Neither am I afraid of your drinking one or two glasses of wine *after dinner*, though I do not wish you to make a rule of that sort, and it is clearly better to abstain altogether than by taking a little to endanger the taking much. Eat freely of animal food at dinner, but confine yourself to one dish. Avoid all high seasonings of every kind. Be cautious in the use of butter, oil, or vegetable acids." . . . "Where the gout has continued long, the life is far advanced, the strength much impaired, the doctrine of abstemiousness is to be applied with great caution; and if the frame be much emaciated, it is not to be applied at all. In such circumstances a cordial regimen is the most safe, especially if it corresponds with long-established habits, because it is now too late to aim at abating the violence of the disease, and the object is to keep up the strength under it. Abstemiousness might suddenly lower this, and bring on complaints for which the gout would be ill exchanged" (*Advice to a Gouty Patient.* By Dr. Currie, of Liverpool. 1790.)

"A slight excess of any kind, whether in diet or exercise, will excite the disease in those predisposed to it. When the train is laid an additional glass of claret may have acted as the match, but in all such cases the explosion would have equally taken place had, instead of claret, some other exciting cause fired it." —(Dr. Paris, 1837.)

THE TREATMENT OF GOUT IN ITS VARIOUS FORMS.

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GOUT is a disease which is due to faulty metabolism, probably both intestinal and hepatic, as the result of which certain poisons (possibly the purins and other bodies, but of which we at present know but very little) are produced and lead to an auto-intoxication, which is an early factor in the development of the gouty condition. This auto-intoxication coincides with or is followed by, in the majority of cases, a deposition of sodium biurate in certain of the joints or tissues, which constitutes the climax of the gouty attack. I cannot but think that with our increasing knowledge and experience of the disease, uric acid and its salts will in all probability have to be relegated to a position of subsidiary importance in the pathogenesis of gout. The joint-manifestations are probably dependent upon much more general and much larger conditions than a mere excess of uric acid in the blood. The deposition of sodium biurate is possibly merely the sign of the disease, not the essence of it.

As regards the treatment of gout, in the first place it should be borne in mind that no routine treatment can be adopted which is suitable to all cases. The nutritional condition of the patient, his habits, surroundings, and mode of life, constitute factors that must necessarily modify the treatment of individual cases, and with gout, as with so many other diseases, it will be found that each individual case requires separate study and frequently special treatment. Quite apart from the treatment of an attack of gout, which is a comparatively simple and easy matter, must be considered the treatment of the condition or conditions which led up to the attack. In connection with this point it must be remembered that the gouty individual is one whose general metabolism is unstable, and that this instability may be present in one or more of the great physiological systems—the digestive, the nervous, the circulatory, &c. The question which of these systems is primarily and mainly at fault should always be a matter for patient investigation, and one must then endeavour to improve the metabolism of that system by suitable medicinal, dietetic, and hygienic treatment.

The treatment of gout should have for its aim the following objects :—(1) The treatment of the gouty paroxysm in cases of acute gout, and the relief of the pain as speedily as possible; (2) the treatment of the subacute or chronic condition and the prevention of the recurrence of an attack, which may be effected by the promotion of the elimination of uric acid and the other purin-bodies, by checking any excessive formation of these bodies that occurs in some subjects, and by careful attention to diet and general hygiene; and (3) the treatment of the affected joint or joints with the object of removing the uratic deposits and of preventing permanent deformity.

In all cases of gout a very careful examination of the urine should be made in order to ascertain whether there is any organic kidney-affection. The indications of such affection of the kidneys are the existence of a certain amount of polyuria, a low specific gravity of the urine—usually from 1007 to 1016 —the presence of a small quantity of albumen (which, however, may disappear for some time and then re-appear), the presence of a few granular casts if a careful microscopical examination is made after centrifuging the urine, and a diminished daily excretion of uric acid and generally of urea. It is most important carefully to examine the urine for traces of albumen, and for the presence of casts. For the latter purpose the centrifugal machine should be used, as the casts, when present, usually occur in but small numbers, and are otherwise very slow to settle.

THE TREATMENT OF ACUTE GOUT.

For the treatment of the gouty paroxysm the limb should be placed in the horizontal position, or slightly elevated above the level of the body, and a cradle should be arranged so as to take the weight of the bed-clothes off the affected part. To alleviate the severe pain felt in the affected joint, warm packs should be arranged round it, consisting of cotton-wool saturated with a soothing lotion, and then lightly covered with oil-silk.

I have found the following lotion most useful in relieving the local pain :—

Sodii Carb.	℥ iv.
Linim. belladonnæ	ʒ ii.
Tinct. opii	℥ iss.
Aq. ad	℥ viij.

A small portion of the lotion should be mixed with an equal quantity of hot water, and then poured on cotton-wool previously arranged round the joint. The pack should be changed every four hours. In connection with the acute paroxysm no attempt at local depletion—such as the application of leeches to the inflamed joint, blistering, or incisions —should on any account be made, owing to the great liability of thereby extending the inflammatory condition, and so producing subsequent ankylosis or deformity.

For the internal treatment of acute gout colchicum is one of the most valuable drugs that we possess. It should be especially used for acute gout, and for subacute attacks supervening on chronic gout. If it be used continuously, tolerance is apt to be acquired, and then the drug ceases to act. At the commencement a large dose of thirty to forty minims of colchicum wine should be given, followed by a mixture containing in each dose ten to twenty minims of the wine with from forty to sixty grains of citrate of potassium, which should be administered three times a day. The citrate of potassium, which is given for its combined properties of acting as a diuretic and of diminishing the acidity of the urine, may, if desired, be given as an effervescing mixture, using thirty grains of potassium bicarbonate to twenty grains of citric acid. Colchicum reduces the gouty inflammation, relieves the pain, and shortens the attack. It should only be taken under medical advice, and should never be given in such doses as to produce extreme depression : after the inflammation of an acute attack has subsided the doses of colchicum should be gradually diminished until it is left off.

From three to four grains of "blue pill" should be given the first night, followed by a dose of Epsom salts in the morning. Mercury should be given only in sufficient doses to produce its cholagogue effect, as owing to the possibly defective action of the kidneys the mercury absorbed into the general system may be eliminated with great difficulty. In my opinion it is advisable in the treatment of gouty patients in the acute or subacute stages to avoid the use of saline purgatives owing their efficacy to salts of sodium, on account of the power possessed by all sodium-salts of diminishing the solubility of sodium biurate. In the employment of purgatives for gouty patients the great object is not to produce powerful purgation, but to relieve portal congestion, since a congested condition of the liver means that excessive quantities of glycocine and purin-bodies remain in the circulation, with the consequent production of an excessive quantity of uric acid. A pill containing either two grains of euonymin or a quarter of a grain of podophyllin, combined with a grain of extract of hyoscyamus and a grain and a half of the compound extract of colocynth, will, in many cases, be found to be very useful.

If the pain of an acute attack of gout is so severe as to prevent sleep, chloral, sulphonal, or trional may be given, or a full dose of extract of hyoscyamus given with blue pill at night will, in some cases, act as a very useful anodyne. The administration of opium or morphine should, if possible, be avoided owing to the risk of its deficient elimination, and also on account of its diminishing the amount of urine, and its tendency to derange digestion and to check hepatic metabolism.

DIET IN ACUTE GOUT.

During an attack of acute gout a diet must be given which shall tend to check the abnormal metabolism of the gastro-intestinal tract and of the liver, which shall be non-irritating to the kidneys, and which shall be one that diminishes as far as possible the production of the purin-bodies. For the first day or two of an acute attack the patient should be restricted to a milk-diet, which may consist of milk, bread-and-milk, and tea made with boiling milk instead of with water. Weak tea with cold toast thinly buttered may also be taken. The free drinking of hot or cold water, of Salutaris water, or of some mineral water free from sodium-salts should be encouraged. The milk-diet should be continued until the acute inflammation is subsiding, which stage is indicated by the lessening of the pain, and by the pitting on pressure of the affected parts. No alcohol in any form should be given during this stage, unless there are strong reasons for its administration, such as a weak action of the heart and a feeble, irregular pulse, when a little well-matured whiskey or brandy, diluted with Salutaris water, will prove the best form of alcohol. Beef-tea and any of the meat-extracts or essences should be avoided at all times by gouty patients, owing to the tendency they have to irritate the kidneys, and to introduce into the circulation waste nitrogenous bodies. With the subsidence of the acute attack the patient may return to a more liberal diet, but care should be taken to avoid anything indigestible. The dietary suitable for gouty subjects after the acute attack has subsided will be considered later on.

THE TREATMENT OF SUBACUTE AND CHRONIC GOUT.

Means of Checking the Excessive Formation of the Purin-Bodies.—These consist in careful attention to diet and regimen, in the promotion of the metabolism of the liver, so as to check the excessive production of the antecedents of uric acid, and in the relief of congestion of the portal system, which can be effected by keeping the bowels open at least once a day. In addition to colchicum, which may be given in small doses, guaiacum may very usefully be administered as an alterative which stimulates the metabolism of the liver, and also affords relief to the portal system. From five to ten grains of guaiacum resin should be given in cachets two or three times a day, according to the effect on the bowels, since guaiacum sometimes acts as a laxative. The method of administering the powdered guaiacum resin in cachets is far preferable to giving the tincture of guaiacum in a mixture, as in the latter form a nauseous medicine is produced, and the precipitated resin tends to cling obstinately to the tongue and fauces. In cases of chronic gout the colchicum may very conveniently be administered in the form of a pill, given three times a day, containing one-sixtieth of a grain of colchicine, combined with one quarter of a grain of extract of nux vomica and one grain of extract of gentian. Colchicine, however, should not be given in cases of marked interstitial nephritis, as in such cases a fatal result has been known to follow its administration in medicinal doses. If constipation occur, a sulphur-and-guaiacum tablet, or a dose of compound liquorice powder, should be administered at night. An occasional dose of blue pill and euonymin, followed by a purge of Epsom salts, will be found useful. If the patient is suffering from atony and debility of the stomach, nux vomica or strychnine may be given with potassium citrate. Iron-preparations are not as a rule well tolerated by the gouty, but if anæmia is present the citrate of iron and ammonium or the carbonate of iron will be found the best to administer.

Means of Promoting the Elimination of Uric Acid and other Purin-Bodies.—This may be effected by medicinal treatment, and by diet and regimen. Citrate or bicarbonate of potassium should be employed as a diuretic, which increases the volume of the urine and at the same time diminishes its acidity. The use of the potassium-salt may with advantage be pushed until moderate alkalinity of the urine is produced, as by such means the quadriurates are rendered more soluble and more stable than they are in acid urine, and so the tendency to the deposition of uric acid or sodium biurate in the kidney-tissues is removed. Free diuresis should also be encouraged by the drinking of sufficient quantities of water. A patient suffering from gout should avoid the excessive use of common salt at table, owing to the power it possesses of diminishing the solubility of sodium biurate, and thereby hastening the precipitation of that body.

The Use of Alkalies and the Salts of Alkalies.—Of the potassium-salts used in the treatment of gout the citrate and the bicarbonate are the two most commonly employed. As to the beneficial effects of employing a potassium-salt in conjunction with colchicum in the treatment of acute and subacute gout I am fully assured, and my experience is that of the various potassium-salts the citrate is the most useful. If given in sufficiently large doses it tends, by its conversion in the kidneys into the carbonate, to diminish the acidity of the urine, which is generally high in connection with the gouty paroxysm, while at the same time it increases the solvent power of the urine for the uric-acid salts, and so assists their elimination. In addition, as I have experimentally shown, the presence of a potassium-salt both delays and inhibits the conversion of the soluble gelatinous sodium biurate, which is the form in which the biurate is first present in the blood, into the comparatively insoluble crystalline biurate. In this way the deposition of the latter in the tissues is inhibited, and so further time for the elimination of the biurate is offered. I do not however, wish to contend that the above-mentioned are the only explanations of the beneficial action of potassium-salts in the treatment of gout. In addition they have their uses in the gouty state on account of their stimulating action upon metabolism, of their remedial action upon the gastric and hepatic functions, and of their diuretic effect.

As regards the use of sodium-salts they are certainly beneficial in some gouty conditions, but since they are directly detrimental to the removal of gouty deposits I am of opinion that those mineral waters should be avoided which owe their activity to those salts *when the removal of gouty deposits is the main object of treatment.* But in cases of sluggish action of the liver, of gastro-intestinal catarrh and torpor, of gouty dyspepsia, and of other forms of irregular gout where there are no appreciable uratic deposits in the joints, mineral waters containing sodium-salts are undoubtedly beneficial, owing to the action of those salts as hepatic and gastro-intestinal stimulants.

As regards the use of lithium-salts in the treatment of gout, my opinion is that they are not so useful as the potassium- and sodium-salts. The lithium-salts have not the same inhibiting effect on the conversion of gelatinous sodium biurate into the crystalline form as the potassium-salts have, while at the same time they have no better solvent effect on gouty deposits. The great objections, however, to the use of the lithium-salts is their greater toxicity and depressing action on the heart as compared with the potassium-salts. They consequently have to be given in such small doses that I am very doubtful as to whether in such doses they possess any remedial effect at all. On the other hand, I constantly meet with patients suffering from cardiac depression as the result of the excessive and continued consumption of lithia-tablets, which are so persistently, so speciously, and so wrongly vaunted as curative of gout.

In the treatment of the gouty state associated with disturbance of the functions of the liver the most important consideration is the restoration of that organ to its normal state of activity, and here the alkaline sodium-salts are especially useful. There is no better treatment at the outset than a dose of blue pill or calomel at night, followed by a dose of Epsom salts or Carlsbad salts in the morning. Subsequently a pill containing a small dose of "blue pill" or calomel combined with euonymin and colocynth will be found most useful. In such cases of gouty hepatic inadequacy a mixture which I have found most beneficial as regards its stimulating effect on the metabolism of the liver, and also of the gastro-intestinal tract, is one containing sodium bicarbonate, gentian, and nux vomica, taken a quarter of an hour before meals.

Further Treatment of Chronic Gout.—The enlargement and tenderness of the gouty joints is due to two causes—the deposition of sodium biurate in the cartilages and fibrous structures, and a chronic inflammatory thickening of the fibrous tissues. For the reduction of this last-mentioned thickening, as well as for painful gout of the sole of the foot, and for gouty neuralgic affections, iodide of potassium given internally is a useful remedy. This drug should not, however, be regarded as a solvent of gouty deposits, and it is contra-indicated if advanced kidney-disease exists. It should be given in doses of five to ten grains three times a day, and may usefully be combined with from five to ten minims of tincture of iodine.

THE LOCAL TREATMENT OF GOUTY JOINTS.

If much swelling of a joint persists, the limb should be elevated as much as possible, and a light flannel bandage applied to the joint. If the œdema persists, the hot douche followed by sponging with a cold strong solution of common salt will be found serviceable. The application of the so-called solvents of uric acid externally to affected joints is useless, as they are not solvents of sodium biurate. Careful massage and gentle exercise of the stiffened joints should be employed, but only when convalescence is fairly established. For the stiffness and thickening of joints, careful rubbing with Iodide of Potassium and Soap Liniment or with the Compound Camphor Liniment may be resorted to. The thermal baths of Bath, Buxton, Harrogate, Strathpeffer, Llandrindod Wells, Aix-les-Bains, and other spas, and mud-baths, are useful in the treatment of cases of chronic articular gout. Treatment by means of baths should, however, be avoided by patients suffering from acute gout, by elderly patients, and by those suffering from any serious cardiac affection.

BATHS AND MASSAGE.

A sedentary life and deficient exercise conduce to gout, owing to a lowering of general metabolism, to depressed vigour of blood-circulation, and to impairment of movement of the lymph in the lymph-channels. Since the lymph-circulation is mainly carried on by muscular movements and respiration, the beneficial influence of massage and muscular movements on many gouty subjects becomes readily intelligible. Massage should never be resorted to in cases of acute gout, as such treatment not only aggravates the disease at that stage, but also causes severe pain; it should be reserved for the more chronic cases. Massage produces an increase in the amount of blood and lymph passing through the tissues concerned, both during massage and after its cessation. This increased circulation improves the nutrition of the affected tissues, promotes absorption of deposits, and restores physiological activity. In cases of subacute or chronic gout, where the joints remain swollen and œdematous and are the seat of considerable deposits, much benefit is frequently derived from massage and galvanism. The affected joints should be massaged, each one for a few minutes, and then galvanism (five to ten milliampères) applied to each joint for a few minutes with the negative pole over the affected region. The galvanism is then to be followed by a few minutes' massage. Under this combined treatment the œdema and the deposits frequently disappear rapidly. Probably the beneficial effects are due mainly to the increased circulation of blood and lymph induced, and the consequent absorption that takes place. I have also found in many cases that a decidedly beneficial influence on gouty joints is produced by radiant-heat baths, followed by electrical treatment in the form of cataphoresis to the affected joints.

Radiant-heat and superheated-air baths increase the oxidative processes within the body, as is shown by the increased elimination of carbon dioxide from the lungs, and also by the increased metabolism of the body in general. They also stimulate the circulation both of the blood and lymph in the affected joints and so lead to improved nutrition of the parts. This curative action undoubtedly continues after the treatment has been left off. Such treatment therefore is better given intermittently—say, six baths on alternate days; then intermit for two or three weeks and so on. These baths improve the atrophic condition of the muscles. They cause a temporary elevation of the body-temperature, marked reddening of the skin of the part treated, profuse local or even general perspiration, quickened pulse, lowered arterial tension, and generally considerable amelioration of the pain, and in some cases complete disappearance of it for a time. Radiant heat has a greater penetrative effect than other forms of heat, and, in my opinion, the effect is more stimulant. In cases of acute or subacute gout the pain, as a rule, recurs at varying intervals after a bath, but usually with diminished severity; and in favourable cases a progressive reduction of the pain occurs after the use of subsequent baths. Undoubtedly many cases of chronic gout do not show much improvement after the use of radiant-heat or superheated-air baths, and I have frequently experienced great difficulty in selecting the cases most likely to be benefited by it. As a general rule my experience has been that cases of chronic gout of long standing, with considerable deposits in the joints, do not derive much benefit from these baths. For such cases undoubtedly much more good can be done by the employment of vapour-baths, followed by massage of the affected joints, a method of treatment which is frequently most useful in producing softening and absorption of the deposits. It should, however, be borne in mind that radiant-heat baths seem to set up improved circulatory and trophic changes in the joints, which apparently are maintained for a prolonged period of time after the baths have been discontinued. Certainly I have seen in several cases, after a course of twelve or eighteen radiant-heat baths had resulted in but slight improvement, and the baths had been abandoned in despair, a progressive improvement maintained for weeks and even months after the discontinuance of the baths, an improvement which in some cases has progressed to more or less complete cure. If only one limb is the seat of gout, the question arises as to whether that limb should be locally treated by being placed in a small specially-constructed bath, or whether the "entire body" bath should be used. My experience is that the "whole body" bath is in all cases the most useful, with, in the case of the radiant-heat bath, an extra localisation of the heat- and light-rays on the affected part. That means, according to my experience, that the more extensive the surface to which the heat- and light-rays are applied the better is the result. When the ordinary radiant-heat or superheated-air baths are not obtainable, very good results may be obtained at home by the use of an ordinary blanket-tent with a small opening at the top to let out the hot air saturated with moisture. The hot air is supplied by a ring bunsen gas-burner, or by a large spirit-lamp with a flue passing through an opening in the blanket at the foot of the tent.

Cataphoresis is useful in many cases of chronic gout with considerable deposits in the joints, and also in many cases of obstinate rheumatoid arthritis. It consists of the electric transfer of fluids through the joints from anode to cathode. The best fluid to use in cases of gout is a solution of potassium bicarbonate. The padded negative electrode should be placed on the back or chest, and the positive electrode, which should be button-shaped and padded, and which is kept wet with the saturated solution, should be applied to the joint. Currents of from 10 to 30 milliampères may be employed. For the treatment of gouty neuritis constant-current baths are usually most beneficial; radiant-heat or superheated-air baths followed by the use of the constant current generally do an equal amount of good. In the acute or subacute stage of gout, or, when a slight attack of gout has just started, I consider the use of the Turkish bath most undesirable. I have known of its employment in such cases being followed by an exacerbation of the attack and extension to joints not at the time affected. This is a point which should be borne in mind by medical men, as many patients on the first appearance of an attack of gout are apt to have immediate recourse to the Turkish bath, and it is well that they should be warned of the danger they thereby incur.

The Treatment of Gouty Eczema.—In the treatment of this form of irregular gout special attention should be given to two details. One is to see that the bowels are freely opened, which at the outset may be secured by the administration of blue pill or calomel followed by a saline; the other point is that entire abstention from alcohol is most desirable, at all events during the treatment and persistence of the eczema. It is best that any form of alcohol should be abstained from, but the prohibition applies more especially, in my experience, to the red wines. I have met with several cases occurring among gouty individuals past the middle age of life in whom two or three glasses of claret or burgundy will in the course of a few hours cause the development of an eczema. During the irritative stage of dry gouty eczema I have found the application of a lotion consisting of liquor plumbi subacetatis ʒj, liquor carbonis detergens ʒj, aqua sambuci ad Oj, most soothing, especially if followed by the use of a simple dusting-powder such as cimolite powder. For the acute moist type of eczema a similar lotion, but with a preparation of opium replacing the tar-solution, is advisable. In cases of gouty pruritus, or, as it is sometimes termed, latent gouty eczema, the severe itching is frequently relieved by the use of carbolic-acid lotion. When the eczema is in a chronic condition much benefit is usually experienced from immersion in sulphur-baths, such as those of Harrogate, Strathpeffer, Aix-les-Bains, &c. After the bath the skin should be carefully dried, and a dusting-powder such as cimolite powder freely applied. In cases of gouty eczema and gouty pruritus a careful dietary must be enforced, care being taken to forbid all articles which the experience of the patient in the past has shown to produce dyspepsia. Persons who are subject to attacks of gouty eczema should avoid such acid fruits as strawberries, gooseberries, apples and pineapples; rhubarb also should be excluded from the dietary. As regards the medicinal treatment of gouty eczema, my experience is that it is not necessary to give the ordinary anti-gout remedies, such as colchicum, &c. It is much more important to treat the dyspepsia and the catarrhal condition of the gastro-intestinal tract, which are generally present as associated or causative conditions, by the administration of subcarbonate of bismuth with the bicarbonate of sodium or the bicarbonate of potassium.

Treatment of Insomnia.—Insomnia is a fairly common symptom in gouty patients. As a rule, it is not complete, but consists of restlessness, interspersed with varying intervals of light or broken slumber. In such cases attention should always be paid to the condition of the patient's pulse, and to the possible existence of albumen in the urine. If, as is frequently the case, the pulse is found to be of high tension, the administration of blue pill or calomel, followed by a saline purge, will probably prove most beneficial.

With regard to the use of tobacco, the majority of gouty patients may be permitted a moderate indulgence in smoking, but it should be avoided entirely by those who suffer from paroxysmal attacks of the gouty heart.

PREVENTIVE TREATMENT OF GOUT.

I have now had a considerable experience of the prophylactic effects of guaiacum resin, and I must confess that I know of no drug which is more useful in the preventive treatment of gout. Its action is probably due to its stimulating effect on hepatic metabolism, thereby increasing, as it undoubtedly does, the elimination of uric acid. The form in which I prefer to give it, is that of the powdered resin in cachets, commencing with doses of 5 grains three times a day after meals, and gradually increasing the dose to one of 10 or 12 grains. In this form it can be taken without any discomfort to the patient, whereas if administered in the form of the tincture in a mixture a most nauseous medicine results.

Quinic acid is a comparatively new drug, which in various forms of combination has been put forward as possessing some beneficial action in the treatment of gout. It diminishes the output of uric acid in the urine, but simultaneously increases the excretion of hippuric acid. My experience of the different combinations of quinic acid is somewhat limited, but so far I am inclined to think that they are of decided use in the treatment of certain forms of chronic gout. The administration of fruits frequently reduces uric-acid excretion, a result which has been attributed to the effect of quinic acid. Gowland Hopkins considers that this diminution is due to interruption of some synthetic process, rather than to a mere effect on excretion.

After convalescence, as much exercise as possible, short of fatigue and discomfort, should be taken in the open air. Cycling is an excellent exercise for the gouty, since it furnishes good muscular movement in the open air without the gouty joints having to bear the weight of the body.

DIET.

No hard-and-fast lines as to dietary can be laid down in the treatment of gout. Each individual must be carefully considered as regards his habit of body, his capacity for the digestion of different articles of food, the amount of exercise he is able to take, and the nature of his work. Derangements of the gastro-intestinal tract constitute a most important factor in the development of acute, chronic, and irregular gout. It is, therefore, of the utmost importance to secure and maintain a healthy condition of the gastro-intestinal mucous membrane, and a normal daily evacuation, in order to guard against auto-intoxication, which is undoubtedly an early factor in the development of the gouty condition. The individual who is subject to gouty attacks can certainly diminish the number and severity of the attacks, and in many cases can prevent their recurrence, by careful attention to diet, to the quality and the quantity of fluid taken, to exercise, and to a sufficient daily action of the bowels. It is my custom to question closely each gouty patient that I see, not only as to the nature of the beverages taken, but also as to their amount; and my general experience is that the great majority of individuals suffering from gout take an insufficient quantity of water to drink. Consequently there is an insufficient flushing of the liver, kidneys and other organs and tissues, and therefore imperfect removal of waste and toxic products. More especially does one find this insufficient consumption of fluid among female patients, in many cases due to the absurd and erroneous belief that a diminution in the amount of fluid taken tends to keep down the body-weight and to prevent the occurrence of obesity. Taking from my case-books ten consecutive cases of gout occurring in ladies whom I carefully questioned as to the amount of fluid consumed per diem, I find that amongst these ten the amount averaged only 26 fluid ounces; this included all fluid, whether taken as water, tea, coffee, soup, wine, ale, &c. The amount is obviously insufficient for the proper flushing of the system. For the treatment, as well as for the prevention, of the gouty condition the free consumption of water apart from meals is most desirable.

As regards the question of meat it must be remembered on the one hand that animal foods constitute to the majority of people the most attractive and appetising forms of diet, and are therefore likely to be taken in excess; hence the necessity for limiting the amount to be taken. But on the other hand it must be borne in mind that it is most desirable to increase the combustion and the oxidative powers within the tissues. In my opinion it is absolutely erroneous to exclude from the dietary of the gouty such articles as meat, fish, and tea because they are assumed to contain uric acid. I have yet to learn that any uric acid is present in these articles of diet, since the so-called estimations of uric acid in them are not, as I have previously pointed out, estimations of uric acid at all. Moreover, the deduction is an erroneous one that because uric acid is a nitrogenous body, it must therefore be directly derived from nitrogenous constituents of the food, the consumption of which must consequently be avoided. Even if uric acid were present in the articles of food referred to, it would not alter my opinion as to their suitability, considering that they have stood the test of so prolonged a trial. Yet there are some who do not hesitate to call these articles of food, which are so exclusively consumed, poisons, mainly, as far as I can gather, because these foods do not happen to agree with themselves. It is true that there are a few persons whom, without any intention of being offensive, I should describe as physiological degenerates, who find that such articles of food do not agree with them, but it is illogical to argue from such a premiss that therefore they are unsuited to the great majority whose digestive functions are more happily regulated.

The diet of gouty patients should be simple, that is, the meals should not be made up of too many articles. Simplicity of food means facility of digestion. Certainly meat, even red meat, should not be excluded from the diet. No class of food-stuff is so productive in energy as animal food; and as most cases of chronic gout are suffering from lowered vitality and want of tone, animal food, at all events in moderate quantity, is distinctly indicated. My experience supports the truth of this view, as I advise in the great majority of cases of chronic gout the taking of at least one meat-meal a day. The exclusion of any article of diet, or of any class of food, without taking into account the surroundings of the case and the peculiarities of the individual is unscientific. Those articles of diet that are known in the individual to favour intestinal fermentation and putrefaction should certainly be avoided, and it may be taken, I think, as a general rule that a sense of discomfort after a meal indicates that some article or articles of food have been taken which are not beneficial to the individual in his present condition. I attach great importance in such cases to the reduction of the starchy articles of food, but not to the total exclusion of, what I believe to be comparatively harmless, the potato. It is remarkable how frequently one hears from gouty patients the emphatic statement, "I never eat potatoes." I must confess that I do not know of any good and sufficient reason for this wholesale condemnation of this common article of diet. Undoubtedly amongst those gouty patients who suffer from an inability to digest starchy articles of diet—in other words, who suffer from amylaceous dyspepsia—a reduction for the time in the amount of starchy foods taken, including potatoes, is desirable; but the recognition of the existence of amylaceous dyspepsia is a fairly easy matter, and when present it can be suitably treated. Certainly those who are gouty and fat should be very sparing in the use of potatoes, as of other carbohydrate forms of food. I wish, however, to protest against the too general exclusion from the food of the gouty of so common and useful an article of diet as the potato. Equally wrong, in my opinion, is the total exclusion of sugar from the dietary of all gouty individuals. Undoubtedly in certain individuals sugar may do harm, as in the cases of gouty persons who are fat, or who suffer from glycosuria, or who are prone to attacks of eczema, and in such it should be cut off; but that is no reason for the exclusion of it from the dietary of all gouty patients, especially those who are at the same time gouty and thin. I know of many gouty individuals who take sugar with absolute impunity. Some gouty subjects undoubtedly digest very badly all starchy articles of diet, and in such fats may well take the place of starches. Fat bacon, properly cooked, is generally well digested by gouty individuals.

A fair proportion of vegetable food should be taken with two meals each day. The choice of vegetables will depend upon the digestive capacity of the patient; but, excepting the potato, as a rule those vegetables that grow above ground are preferable to root-vegetables.

Stated as a general principle, a person who is subject to gout is better without alcohol in any form. There are, however, some who require a little alcohol, either to aid digestion or to enable them to get through their work, and here I am entirely in accord with the advice given by Goodhart, that, if a man requires any stimulant at all, it is a matter he must decide by experiment for himself, for no medical man can tell him. If alcohol is necessary or desirable, the form in which it is to be taken is frequently a matter which the patient can decide better than the medical man; but I would insist upon the importance of definitely limiting the amount to be taken, and of restricting its consumption absolutely to meals. Some patients find that a little whiskey or brandy suits them best, others find a light still moselle preferable, while a few, but in my opinion only a very limited number, find that a light claret agrees best with them. Champagne is a wine which is seldom suited to the gouty, especially if taken daily. Rough cider, that is the completely fermented apple-juice, taken in moderation, agrees well with most gouty subjects, but the bottled or champagne cider, which is imperfectly fermented, should never be used by gouty individuals.

As previously stated, as little complexity as is possible in the meals is the main desideratum in the dietary of the gouty, and in a few intractable cases of chronic gout it may even become necessary to reduce the dietary for a time to the simplest possible condition, namely, to two articles of food—lean meat and water. There are a few cases of *chronic* gout which undoubtedly improve and even recover on an exclusive diet of red meat and hot water. These are generally cases of chronic gouty arthritis which have failed to yield to the ordinary methods of treatment, and which are accompanied by dyspepsia, flatulence, acid eructations, pyrosis, and offensive stools. I have successfully treated a few such carefully-selected cases of chronic gout by the employment of this, the so-called "Salisbury" treatment. It is essential, before placing a patient on such diet, that the urine should be carefully examined, as any advanced condition of kidney-disease contra-indicates the employment of such a dietary. If the evidence of kidney-derangement is only slight, the adoption of the dietary is not contra-indicated; but the urine must be carefully examined every two or three days, as any considerable increase in the albuminuria would at once be an indication for the discontinuance of this special diet. Gouty patients suffering from organic heart-disease with any failure of compensation should never be placed on this dietary. The dietary consists in the patient drinking from three to five pints of hot water daily, the water being taken from one hour to one hour and a half before each meal, and half an hour before retiring to rest, and from two to four pounds of beefsteak daily. The meat should be freed from fat, gristle, and connective tissue, thoroughly minced, mixed with a little water, and then warmed through with gentle heat until it becomes brown in colour. A little salt and pepper may be added, and the meat eaten in this form or made up into cakes and cooked on the grill. Later on in the treatment part of the steak may be taken grilled, or a grilled lean mutton-chop may be substituted for one of the daily meals. The course of treatment should last for from four to twelve weeks, after which a gradual return to ordinary diet should be made.

THE USES OF MINERAL WATERS IN THE TREATMENT OF GOUT.

The value of a given mineral water in the treatment of gout depends greatly on the main object with which it is taken. For instance, it may be taken to remove gouty deposits, or to stimulate the action of a sluggish liver and to relieve portal congestion, or for the treatment of gouty dyspepsia, or to relieve the bowels in cases of torpor and gastro-intestinal catarrh, or to act on the kidneys, or to relieve gouty affections of the skin. Now it is manifest that any one mineral water is not likely to produce all these effects, and it is also obviously conceivable that a mineral water which might be most useful to effect one of these purposes might prove most injurious if employed to effect another. No doubt considerable error has arisen from indiscriminately sending gouty patients to a particular spa without giving due consideration to the question as to whether the water of that spa is suitable for the treatment of the specific gouty disorder from which the patient is suffering. Moreover, it is well to bear in mind that a patient should not be sent to a spa during the acute stage of gout, nor if suffering from marked organic disease of the heart or kidneys.

The use of a mineral water, so far as its employment with the object of removing gouty deposits is concerned, lies solely in its watery constituent, and does not in any way depend on the mineral constituent dissolved in it. As a matter of fact the salts dissolved in some of the natural mineral waters are directly harmful in gout, both by encouraging deposition of the sodium biurate and by checking solution of the gouty deposits. The flushing of the system of a gouty patient with abundant quantities of water is undoubtedly beneficial, since it dilutes the blood for the time, and so tends to prevent uratic precipitation, and at the same time promotes diuresis and encourages elimination. The question, however, naturally arises whether, if the water of a mineral water be its only beneficial constituent for effecting the removal of gouty deposits, the sending of gouty patients to spas for such a purpose presents any advantages over their drinking ordinary water at home. If the conditions of the life of the patient at home and at a spa were the same, there would be no such advantage; but among the special benefits to be derived from residence at a spa must be reckoned the almost undistracted attention that is given by the patient to treatment, the careful dieting that is frequently observed, the change of surroundings, the absence of business or home worries, and the opportunities for the use of thermal baths for the external treatment of articular gout. It should, however, be borne in mind that owing to the undoubted fact that sodium-salts are directly detrimental to the removal of gouty deposits, those springs should be avoided which owe their activity to those salts, *when the removal of the deposits is the main object to be attained.* The springs which contain no sodium-salts, or traces only, are those suitable for such cases. I wish it to be clearly understood that I am by no means condemning the very proper uses to which mineral waters containing sodium-salts can be put for the treatment of many gouty affections of the viscera and other structures, but I wish to emphasise the point that when the system is flushed with a mineral water with the object of dissolving and removing gouty deposits, then it is undoubtedly advisable to select a water as free as possible from sodium-salts. On the other hand, in cases of sluggish action of the liver, of gastro-intestinal catarrh and torpor, of gouty dyspepsia, and of other forms of irregular gout where there are no appreciable uratic deposits in the joints, mineral waters containing sodium-salts are undoubtedly beneficial, owing to the action of those salts as hepatic and gastro-intestinal stimulants.

CLIMATIC TREATMENT.

A fairly bracing air with a low relative humidity is, in my experience, the most suitable for the gouty. High mountain situations and valleys where there is an excessive relative humidity of the air are alike unsuited to the gouty. Especially is it desirable to avoid exposure to the cold east and north-east winds which prevail in this country in the early months of spring, and which are so apt to be provocative of what has been called a "chill on the liver," a condition which no doubt is brought about by the chilling effects of these winds on the skin, and a consequent reflex affection of the metabolism of the liver-cells. As a winter resort for the gouty I know of no better climate than that of Egypt, where, at Helwan (Helouan), thermal, sulphurous, and saline waters exist, and excellent baths are obtainable. The air of Helwan is that of the desert; the average winter temperature is 60° F.; the relative humidity from November to April is only 30 to 60 per cent.; while the average rainfall for four consecutive winters was only three-quarters of an inch. For the spring, summer, and autumn months we fortunately have for our selection a large number of health-resorts in this country and on the Continent, the climates of which are well suited to the gouty. My experience is that residence by the sea is not suited to most cases of gout, and this especially applies to cases of gouty eczema.

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BALNEOLOGICAL TREATMENT OF GOUT,

WITH SPECIAL REFERENCE TO BATH.

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THE treatment of gout by the drinking of, and bathing in, certain mineral waters forms one of the most effective weapons in a physician's armamentarium. At the same time one must remember that different waters are most advantageously employed in different stages of the disease, and in different physical states of a patient. While, therefore, broad lines can be laid down as to the suitability of any individual water for a particular case, many things must be taken into account besides idiosyncrasy in ordering a case to a certain spa and for a certain course of treatment. It is with the hope of assisting the medical profession in the choice of a suitable spa that I have written the following notes on the balneological treatment of gout.

It is not my intention to enter into the causation and pathology of the disease, but to assume as a fact that in gout we have to deal, in one case, with an excess of urates in the blood, and in another with a deposition of the urates in the tissues. Uric acid does not exist in the blood in a free state, but may be found there in combination as a urate (quadriurate). In the normal state this is eliminated by the kidneys, but should anything interfere with this elimination, certain changes occur which transform the soluble urate (quadriurate) into biurate of sodium, and in this form it is deposited as an insoluble crystal in the tissues. Our aim in the treatment of gout, therefore, must be, in the first instance, to diminish the tendency of the system to accumulate urates in the body-fluids, and secondly to prevent the deposit of them in the tissues as crystals of sodium biurate. Unfortunately the former tendency is a most persistent one, and one which, although capable of modification, does not seem susceptible of any permanent and radical cure.

Failing to prevent the formation of an excessive proportion of urates, it becomes necessary to endeavour to lessen the percentage of them in the body-fluids, for it has been proved that the larger the proportion of urates present the more likely is there to be a deposition of the crystalline biurate, or, in other words, an attack of gout. If we bear these two principles in mind, it becomes clear how the various mineral waters may help, and in what way their effect may be looked for. The *rationale* of mineral-water treatment, in fact, becomes clear.

As may be judged from the foregoing, the gouty diathesis is quite as important to treat as gout itself, for if we can prevent an excessive quantity of urates accumulating in a person known to have a tendency to gout, it is quite conceivable that actual deposition of the biurate crystals may be prevented, or at least postponed for a great length of time. It is, therefore, of importance to remember that balneological treatment must be repeated at suitable intervals, according to the case, and that, in no case, can one or two courses of waters be looked on as likely to produce a permanent cure. From the nature of the disease this cannot be; but I wish to emphasise the fact, as one so often finds grave disappointment in those who, having once undergone a course of treatment at a spa, think they have done all that is required to keep them free for the rest of their lives. My experience is that when once the formation of an excess of urates has begun, there is a constant fight between the prophylactic and eliminative measures adopted and the formative tendency on the part of the body, and in nine cases out of ten the latter wins.

The two points in treatment to be arrived at are therefore: (1) The prevention of the excessive formation of urates; and (2) if the excessive formation has occurred, the elimination of the excess.

At spas we can assist in preventing the excessive formation, by superintending and inculcating certain rules as to exercise, diet, mode of life, and personal hygiene. Should there be excessive formation or deposition in the tissues, we can, by prescribing certain waters to be drunk and bathed in, assist the elimination of the excess of urates, and diminish, by flushing out the system, the percentage existing in the body-fluids. These are our principal aims.

Gout presents itself in many and various guises, and in practically all of them balneological treatment is of use. It may occur as regular or irregular gout, and in every gradation of acute or chronic form. It may be seen as an acute paroxysm affecting one or more joints, with or without deposits in the cartilages and fibrous structures of the articulation. It may be chronic with deposits in and about the joint-structures, in the ligaments, tendons, and subcutaneous tissues, tendinous sheaths and bursæ, causing much enlargement, crippling, and deformity. It may be seen in irregular forms which may implicate practically every function and system of the body. These irregular forms may be related, but frequently they have no relationship to the more regular attacks, and patients suffering from them may never have a regular gouty paroxysm. The principal systems affected may be the circulatory, nervous, urinary, and respiratory; while the skin also may frequently be affected, as may be the eye. It is thus seen that, in dealing with gout, we have a disease which may give rise to almost any symptom, and affect almost any organ or function.

Bearing all this in mind, and the nature of the disease, it becomes a question what to do with each individual case, and what method of treatment to recommend. This is a difficult question, but when once it is decided that a course of mineral waters is necessary, one is often asked whether drinking the waters at home will not do. Theoretically it should, but we must not forget that drinking the waters is only part of the cure. The waters at most spas are also used in the way of baths and various kinds of douches, which may be supplemented and assisted by various forms of packs, passive movements, and massage, electricity, radiant heat, &c. Moreover, in getting away from home and in taking a holiday, the daily worries are to a great extent left behind, and the patient has the advantage of change of air and scene, superintendence of his diet and mode of life, and plenty of opportunities for outdoor exercise. Another great point in going to a spa is that it would not be possible for anyone going about their ordinary avocations to drink large quantities of water every day at stated intervals, nor to submit to a series of baths of a more or less trying character. Such a course at any time throws a great strain on the system, and is, without doubt, best carried out at a spa where all the concomitant advantages can also be secured. Empirically we know that gouty persons derive great benefit from a course of mineral-water treatment, for it is a daily observation that patients, afflicted with all forms of the disease, go to spas, and return from them either cured or else greatly improved—and this in cases which have undergone many courses of treatment at home with no result. Therefore we cannot ignore this fact, and we know, from experience, that it cannot be replaced by other modes of treatment.

To explain it scientifically is more difficult. The therapeutic action in drinking any mineral water is supposed to depend partly on the quantity of water taken, partly on the character and quantity of its chief chemical constituents, and partly on its temperature. Hot mineral water acts, therefore, in a three-fold way. It acts to a certain extent by the effect of its constituents, which are rapidly absorbed from the intestinal canal, on many of the glands such as the pancreas, liver, kidneys, &c., as a solvent of various pathological and chemical elements found in the body; as a diluent, flushing and washing out effete material; as an anti-fermentative agent; while by causing a certain over-saturation of many tissues, and, by stimulating the heart's action, it has a decided effect on tissue-changes. Some of these effects are due to its chemical composition and some to the mere fact of its being hot water given in quantity.

When used as a bath, mineral water apparently is not absorbed. The clinical results of immersion in the case of a chlorotic girl prompts one to declare that absorption must occur, but chemical tests declare against it. Bencke and Rohrig have proved that in brine-baths no increase of sodium chloride is excreted by the kidneys, and what is true of this salt must be equally true of other salts. The general consensus of opinion is undoubtedly in favour of non-absorption. Nevertheless it is accepted as true that the skin is capable of absorbing substances that are volatile in the form of gas and vapour. Rohrig proved, by applying finely pulverised water to the skin by force of rubbing, that some absorption took place into the corium, and this of course must be borne in mind when douches accompanied by massage are prescribed. Physiologically hot baths produce an initial vaso-constriction, which quickly passes off, and gives place to a vaso-dilatation, which lasts during the rest of the immersion and then slowly passes off. This superficial vaso-dilatation is associated with an increase in perspiration and in the frequency of respiration. The whole constitutes the reaction of the body to heat, and, as can be seen, the animal mechanism increases the loss of heat to counteract the heating effects of the application. Owing to superficial vaso-dilatation more heat radiates from the body; by increased perspiration the loss of heat by evaporation is augmented; and by increased respiration more heat is given off, and watery vapour expired. In addition, local sedative action on the sensory nerves occurs, which, if prolonged, causes a general sedative action. At the same time the hot water acts as a mild counter-irritant, causing gentle excitation of the cuticle; and this effect is passed by reflex action throughout the body, and is accompanied by increased oxidation. The circulation improves and the tissues receive fresh supplies of blood to reinforce their vital powers. Hot-air and vapour-baths differ only in degree in their action, the greatest amount of perspiration being obtained by hot-air baths. A mineral-water bath therefore acts, partly by causing increased perspiration and expiration of watery fluid from the lungs, with consequent increased imbibition of fluid and flushing of the body-channels; also by its sedative action on the nervous system, and partly by its effect as a stimulant to the whole circulation.

A curious fact has been stated with regard to the precipitation of urates in the laboratory, namely, that the larger the proportion of sodium-salts in the medium the greater is the tendency to precipitation. This is curious, for hitherto the prevailing idea has been that, although not so beneficial as some other alkaline salts, yet sodium-salts were, to a great extent, of use in the treatment of gout; but here it would appear as if they actually do harm. It has also been remarked that those tissues which are most liable to deposits, are those most rich in sodium-salts, and that those tissues which are least rich, are least liable. Thus cartilages and fibrous tissue contain 0·90 and 0·70, respectively, per cent. of sodium-salts, as compared with 0·08 per cent. in muscle. On the other hand Pfeiffer says the best results (clinically) are got with those waters which contain the largest amount of sodium bicarbonate

(Fachengen water). If the laboratory results can be substantiated in the body a remarkable change must occur in our ideas as to the benefit to be derived from certain mineral waters which have been largely used in the treatment of gout.

At many spas one is familiar with the fact that the first effect of treatment may be to bring on an acute gouty paroxysm, which is looked on, in most cases, as being beneficial, for by its means the excess of urates is removed from the body-fluids and deposited in the tissues. This method of cure, being accompanied by much pain and some danger, hardly recommends itself to the thinking physician. It is held that this gouty paroxysm is brought about by the presence of sodium or its salts in the mineral waters drunk, but I have more than a suspicion that these acute attacks are brought on more by the thermal action of the mineral waters, when used externally, than by the action of any of their chemical constituents. This is only my impression, for I have not studied this point closely, but I cannot call to mind any case that developed an acute attack on drinking the waters only, whereas I can remember several which came on only from bathing, the bath being either a simple warm immersion or a vapour-bath. Of course these attacks may have been coïncidences, but I have seen them apparently determined by plain hot-water baths in those who were having no mineral-water treatment at all, and we are all familiar with the fact that soda added to a local bath apparently adds to its sedative action. However, these are points which at present must be left alone. The alkaline and saline waters are usually regarded as giving the best results in gout, as it is thought that the administration of the alkaline carbonates increases the elimination of the urates. But if the theory about the sodium-salts is correct, one is inclined to recommend for the gouty only those waters which contain no soda or at most traces. In the majority of waters which contain little or no soda we find the mineral constituents to consist chiefly of the carbonate or sulphate of lime, and, as lime-salts are, to a great extent, not absorbed into the blood, but pass out of the body without any action, it follows that these mineral waters mainly depend for their action on being drunk freely on an empty stomach.

In considering the clinical results at the different spas we find that in the robust and corpulent the waters of Carlsbad and Marienbad do much good, especially in association with the strict dietetic regimen carried out at these spas. These waters contain large quantities of sodium sulphate, bicarbonate and chloride, hence it is possible that any good derived may be ascribed more to the dietetic regimen combined with the flushing and thermal actions of the waters than to any inherent properties of the waters themselves. In many cases distinct harm is done, at these spas, to old people, especially with a too prolonged course. Again, Vichy, which is a simple alkaline hot water, does much good in the robust cases, but it is not adapted for the weakly and anæmic. The waters of Vals have much the same effect; they contain a very large proportion of sodium carbonate. Of the muriated saline waters those of Harrogate and Homburg are much used in sluggish action of the bowels with portal venosity and congestion of the abdominal organs, but here again we have the objection of the sodium-salts. The sulphur-waters act as cholagogues, thus relieving portal venosity, and are most suitable in gout associated with such a condition. The class of waters which may most suitably be used in all forms of gout appears to belong to the simple thermal or calcareous waters. As most of these contain no sodium, or, at most, traces of it, theoretically they should give the best results, and many have a high and increasing reputation — amongst them are, in this country, the waters of Bath, Buxton, and Strathpeffer; in France, the waters of Contrexéville, Vittel, Aix-les-Bains and Barèges; and in Germany, the waters of Wildbad, Gastein, Pfeffers, and the Sauerling spring at Carlsbad. Taken internally these waters tend to retard or prevent the deposition of the urates. They act on the intestinal mucous membrane as antacids and as slight astringents; and, if taken freely on an empty stomach, they assist the elimination of urates by diluting the blood, and by lowering the percentage present in it. They stimulate the kidneys and relieve the pressure on their excreting powers. Many of them have a great reputation in relieving bladder-irritation and in lessening a tendency to gravel or stone. They are specially suitable in cases of gout amongst the weakly.

Roughly speaking, one can balance certain advantages and disadvantages of the various classes of waters, bearing in mind that, as far as possible, those containing sodium should not be used, if one not containing it can be found to fulfil the other requirements of each individual case. I would therefore in all cases of gout, not associated with marked portal venosity, or the full habit, strongly recommend Bath, Buxton, Strathpeffer, Contrexéville, Vittel, Aix-les-Bains, Barèges, Wildbad, Gastein, and Pfeffers. Should there be much abdominal congestion, with a sluggish liver, it may be considered preferable to send the case to Carlsbad, Marienbad, Tarasp, Elster, Brides-les-Bains, or Franzenbad, or to the slightly less powerful waters of Uriage, Bagnères de Luchon, La Bourboule, Royat, Kissingen, Wiesbaden, or Baden Baden. In delicate cases Ragatz, Teplitz, and Schlangenbad are of use. In America, the most suitable waters are the waters of Saratoga, St. Clair Spring in Michigan, and Caledonian Spring in Canada. These last are alkaline saline waters.

Special complications may require special treatment. Thus gravel and stone receive special benefit at Contrexéville, or, if associated with portal venosity, at Carlsbad. Gouty glycosuria is supposed to derive special benefit at Vichy and Neuenahr, but, from my experience, most mineral waters, generally suitable for the gouty, act as well if associated with proper dietetic supervision. Nervous complications are undoubtedly most benefited at the milder spas, especially the simple thermal ones. If associated with eruptions of the skin the simple thermal or alkaline waters are of much use, as are the sulphur-springs of Harrogate, Strathpeffer, and Aix-la-Chapelle. Some of the mud-baths have also great apparent influence.

There has been much speculation as to the mode of action of mineral waters in gout, and probably the most complete experiments made to determine this were carried out by Pfeiffer of Wiesbaden. He found that in healthy people little or no difference was made in the percentage of urates present in the body-fluids, whereas in gouty patients the percentage was often distinctly lessened, and that instead of circulating free the urates had formed a combination with a base. He is inclined to think that baths, apart from the water-drinking, are of marked benefit.

The actual application of the waters may now be considered. As I am most familiar with the methods carried out at Bath, I will describe the modes of application as carried out there; and it will be found that practically all the best features of treatment elsewhere have been adopted at Bath, while in some cases the technique has been improved upon.

There are two methods of applying the waters, (1) internally, and (2) externally.

Internally the principal use is to drink them, and they may either be drunk as part of a course, with which are associated baths, douches, and other forms of local treatment; or they may form a course by themselves. There are many cases which cannot be bathed for a variety of causes, but which may derive much permanent as well as temporary benefit by simply drinking the waters. The Bath waters, when fresh, are clear and sparkling, without odour, and with a slightly saline chalybeate taste, in no wise unpleasant, and after a time distinctly agreeable. The quantity to be drunk should be divided into two or three portions, one to be taken early in the day, before breakfast, if possible, and the other an hour before lunch or early in the afternoon. In the more robust cases the "before breakfast" glass should always be insisted upon, but with the more delicate this is often impracticable, especially as so many of our cases undergo treatment in the winter months. In any case the water must be taken on an empty stomach. Sometimes the waters are ordered in very large doses, as much as three quarts being given daily, with the result that they act as a purgative. In small doses they tend to constipate slightly. To begin with, the dose should not exceed 4 oz. to 6 oz. at a time, and it should be gradually increased until the full quantity is reached. This, in men, is seldom more than 30 ozs., and in women 20 ozs. To obtain the full benefit the water must be sipped slowly, and gentle exercise must be taken after each dose. At the Grand Pump Room, the water is served at a temperature of 114° F., but in gouty patients more benefit is obtained by raising the temperature of the waters considerably. This is done at the Hetling Pump Room by passing the water over a steam coil, which raises its temperature to as much as 150° F., without any admixture or interference with its inherent properties. It has been found that by thus raising the water's temperature no chemical alteration takes place. The clinical effect of this superheated water is to wash out and dissolve the contents of the stomach, bile-ducts, and upper portions of the small intestines, much more rapidly than occurs when the cooler water at the Grand Pump Room is employed. The immediate effects of the waters when thus exhibited, which are regarded as of good augury, are a glow of warmth in the stomach, an increased appetite, an improvement of the spirits, an augmentation of the saliva, and an increased secretion of urine—this last constituting one of the best indications. They also tend greatly to quench thirst. If, however, the water produces headache, thirst, a dry tongue, and a sense of weight in the stomach, or diminishes rather than improves the appetite, and induces nausea and sickness without any increase in the urinary flow, then it is doing no good. To remedy these symptoms all that may be necessary is to diminish the dose and alter the temperature and the period of the day at which it is taken. Possibly some diuretic taken conjointly with the waters may help, but should they all fail, for a time at least, the use of the waters must be abandoned, as no benefit sufficient to counterbalance the discomfort experienced is likely to be derived. In certain conditions, such as gouty vesical irritation, drinking the water cold is often most beneficial. It can be obtained cold by simply allowing it to stand, but in this case, it will be found to have lost its volatile constituents, and to depend for its action principally on its large proportion of calcium sulphate. In those cases where there is marked portal venosity the Bath waters may be much improved by the addition of some diuretic and laxative ingredient, especially in the early morning dose. The most useful additions being the crystals of citrate of lithium, of the Carlsbad Sprüdel salts, and of those of the Elizabethan spa at Homburg. As an absolute rule it may be laid down that the waters are not to be gulped down, nor taken in a hurry. There is no objection to drinking the waters on the same day as a bath.

The Bath waters are also administered internally by inhalation and as sprays. In gouty conditions of the larynx and bronchi, inhalation of the vapour from the waters has been found to give much relief, but it must be administered with care. The apparatus is the same as that used at Aix, and the gases of the hot waters are inhaled as at Lippspringe. In the "umbrella" room at Bath the hot mineral water gives off its vapour until it fills the room in which the patient sits. The latter at every breath fills his lungs with the vapour, often with marked benefit. After the application great care is required to prevent a chill, and, for this reason, the employment of this form of treatment has been somewhat neglected. With care I have seen much good done. In gouty pharyngeal troubles sprays are also applied, the apparatus used being Siegle's spray, which can be used for hot or cold sprays, with or without the addition of chemical products. Personally I only order the natural waters and always find much good from their use.

From statistics I find that gout, in all its various forms, may be successfully treated by the Bath waters. At the Royal Mineral Water Hospital, over a period of 10 years, 13·9 per cent. of the gouty cases were cured, and 80·7 per cent. were relieved. In taking the percentage of all diseases treated at the hospital, only 10 per cent. were cured, and about 65 per cent. relieved. Therefore the gouty cases show a very good percentage. Under the term "relieved," many leave the hospital quite free from all pain, but, owing to the somewhat strict definition of "cured," they could not be included in that category. In treating gout it is found that a course of treatment tends to remove the various disorders which stand in a more or less close degree of relationship to the actual gouty manifestations, either as antecedents or concomitants, such as gastric irregularities, gastric and intestinal catarrh, acidity, constipation, portal congestion, and urinary disorders. Secondary to the general state are the local changes, such as deposits in and around the joints (chalk and stones), together with inflammatory thickening of the tissues and the consequent articular stiffness, dislocations, contraction of tendons, &c. The mineral waters exert a powerful influence in promoting absorption of these morbid products, whether applied locally or to the body generally, according to the case.

Externally the waters may be applied in various ways.

During the actual gouty paroxysm little can be done by baths beyond the use of the Berthollet Natural Vapour bath to aid the general and internal treatment; but, during the intervals between the paroxysms, much can be done in the way of baths, douches, massage, &c., to effect a cure. As a rule, in the more robust patients, immersion-baths, with or without under-current douches, or possibly vapour-baths followed by hot douches and packs, are indicated; whereas in the atonic form, the milder reclining bath only is used, with the occasional use of local vapour-baths, with possibly massage. It must be remembered that in gout, massage has to be used with care. In many cases it may be applied to all parts except those in which the gouty deposit exists, whereas in others it may be applied even to the parts affected, if great care be taken. During an acute attack it should not be employed. In the more chronic forms, besides vapour-baths, deep and reclining immersion-baths, douches with or without massage, and electricity in many of its forms, are of great use. In gouty cases associated with nerve-pain, the more sedative baths only must be used, such as the Bouillon or plain deep or reclining bath, and without or with electricity. Stimulating douches and massage almost invariably do harm. Associated with a course of baths, sometimes much good is got from a few hot-air baths, either general or local; and, where there is much nerve-pain, the reclining bath associated with electricity may sometimes act in a marvellous way.

As a general rule, the more vigorous the patient the more energetic may the treatment be. Thus the length of immersion may be greater, as may be the temperature, and the heat and time of the pack. It must be remembered that the baths which produce most perspiration are those most trying to the constitution. In prescribing baths no hard-and-fast rules can be laid down, but each case must be judged on its merits.

In the case of nervous subjects or great invalids, it is well to have a servant in attendance, to remain in the dressing-room, if not in the bath-room. An attendant can always be arranged for, but, as the bath-attendants are always busy, it is usually preferable that the patients should, when necessary, bring their own servants to assist with the dressing. At all times a bath should be entered slowly, the water being laved over the upper part of the body before it is fully immersed. In this way the shock, which would otherwise occur, is lessened. This shock or thrill is momentary, and a matter of mere physiological interest. It is found that by wearing a cold towel round the head any tendency to headache is lessened.

When the bottom of the bath is reached the patient should at once sit down on the sedilia provided for the purpose. After the bath there should be a consciousness of increased elasticity and vigour of body, and no feeling of faintness. When attended by marked redness of the skin, flushing of the face, throbbing of the temples and giddiness, the temperature of the bath should be lowered and the duration of the immersion diminished, until in fact both are adapted to the requirements of the case. If, after taking these precautions, similar effects still continue, it will be evident that the use of the baths must be temporarily abandoned.

The temperature of the bath is a matter of great importance. The first two or three should be taken at a temperature varying between 96° and 99° F., preferably the latter. If no ill effect arises, the heat may be gradually raised to 100° or 101° F. More rarely to 103° or 104°. In no case should the latter temperatures be employed without special direction. Experience has proved that more benefit is derived by a continuance of the baths at moderate temperatures than by a short course at higher ranges.

The time for remaining in a bath varies according to the case; ten minutes is a good time to start with, but many full-blooded patients find five minutes long enough for a first bath. As the patient gets accustomed to the treatment, the period may be extended to fifteen or twenty minutes, more rarely for longer periods.

After leaving the bath, the patient, in the majority of cases, will be packed in warm sheets and towels, and left to repose on a couch in the dressing-room for a period varying from five to twenty minutes. The temperature of the pack and the tightness with which the towels are applied varies with the case—the more robust having it hotter, tighter, and for longer than the more delicate. After the time ordered the wraps are laid aside one by one, and dressing slowly proceeded with. It is not, as a rule, desirable for free perspiration to take place after the pack, so that dressing must proceed leisurely; and before leaving the bathing-establishment the patient should remain for twenty or thirty minutes in one of the cooling-rooms provided for that purpose.

After leaving a bath it is advisable that all patients should return home at once in a covered conveyance, and lie down, well covered up, for an hour or more. The best time for a bath is probably in the morning, but many prefer the afternoon as they are then enabled to take more exercise. Any hour will suit, so long as it is not too near a meal. Under ordinary circumstances, three to four baths a week is the usual course. Three to five weeks may be considered a fair duration for the treatment. A short course of less than eight baths is probably pure waste of time, and, by failing to effect a cure in a gouty condition which has probably lasted for years, is apt to bring the waters into disrepute; whereas a steady continuance of their use would, in the majority of cases, give a satisfactory result.

Improvement is often slow in manifesting itself, in the more chronic cases especially, and some patients even do not derive the full advantage of the treatment till a considerable time after its discontinuance.

In most cases the use of the waters should be persevered with, in annual or bi-annual courses, for they tend greatly to retard the natural progress of the disease, and, in many cases, keep the patient entirely free from any gouty manifestations from year's end to year's end—a result probably unattainable by any other form of treatment.

The more common forms of baths employed at Bath for gouty conditions are:—

(1) *The Deep Baths,* which are situated in rooms from 12 to 14 feet long, 8 feet wide, and 11 feet high, and each bath occupies the greater part of the room. Each bath is T-shaped and lined throughout with tiles. Each contains from 800 to 900 gallons of water, with an average depth of about 4 feet 6 inches. The baths are large enough to enable patients to move about, and rings are fixed in the walls for them to hold on to. Should they desire to sit down there are seats all round the sides. The patient descends into the bath by a small flight of steps with rails on either side. Whilst in the bath the patient is almost entirely under water. Should he be unable to walk down the steps, a wooden armchair can be swung from the dressing-room door over the bath, and lowered into it.

The whole apparatus, which is most perfectly under control, is worked by hydraulic pressure. Deep baths are usually given at a temperature of from 96° to 102° F., for ten to twenty-five minutes. With this bath is often associated an under-current douche. This is applied after the bather has already been in the bath for some time. The douche is usually turned on during the last five or seven minutes of the bath, at a temperature of from 100° to 107°, and is delivered through a tube with a nozzle, and, as the water passes through a "mixer" before reaching the hose, its temperature and force can be most accurately regulated. It is directed from a short distance on to the part to be douched at varying pressures. It is most applicable to chronic local gouty affections of the joints, and nothing can exceed the relief and comfort such applications afford. The resistance of the water of the bath necessarily breaks the impact force of the douche, but the douche can be regulated so as to give the required pressure.

(2) *The Reclining Baths,* which contain about 150 gallons of water, are provided with a hose and nozzle, so that an under-current douche can be employed. They are usually employed for those who cannot tolerate a deep bath and may be combined with the under-current douche or with massage. It has been found that a certain number of patients cannot bear the force and volume of the Aix sprays, and in those cases massage in the reclining bath is of much service.

(3) *The Massage Douche Baths* are used principally in the intervals between the gouty paroxysms or where there is much stiffness left after an acute attack. I never order them where there is any chance of setting up fresh mischief or where there is still considerable gouty deposit. The baths are administered in rooms 13 feet 9 inches long by 10 feet 6 inches, with high roofs and with tiled walls. Douches, sprays, and showers are arranged all round the rooms and there is a double water-supply to each, consisting of hot and cold mineral water, so that the temperature can be accurately regulated by means of the "mixer." By this means also the strength of the douches and sprays is regulated. The furniture consists of a wooden table, chair, and stool, all of which are sprayed with hot water before use. During the bath the floor is covered with a layer of water 4 inches deep. If two attendants are

present, each douches the patient with water at the temperature prescribed (usually 99° to 107° F.), whilst applying massage. If only one masseur is ordered, a stream of water is passed through a large rose, and plays over the back or chest of the patient, while the masseur is applying massage. The duration of the bath is usually for 12 to 15 minutes. Towards the end the temperature may be gradually lowered, and the patient may have a cool shower- or needle-bath. After it is over the patient is packed.

(4) *The Needle-baths* or "*douches en circle*" produce a markedly bracing effect, and are usually employed with some other form of bath; they are most useful in nerve-cases, or where there is marked debility. They are applied through six rows of pipes with perforations $1\frac{1}{12}$ inches apart, which are so fine that, with a 2-inch service pipe, five jets cover an area of 8 feet 6 inches. The whole of the body can thus receive the spray at the same time, and the temperature can be simultaneously raised or lowered. Alternating jets of hot or cool water can also be administered.

(5) *Local sprays*, or dry douches, are usually employed when it is undesirable to immerse the patient fully. By means of a hose and nozzle a current of water of varying strength, at a temperature of 110° to 115° F., is sprayed over the part, the rest of the body being covered and kept dry. It is especially useful in chronic local conditions, or where, for some reason, a very delicate patient cannot bathe. Combined with the dry douche, one can order alternating currents of hot and cold water to be played on the patient, and thus have a most stimulating action on the nerves.

(6) *The Berthollet or Natural Vapour bath* may be general or local, and it can be applied with or without a shower-bath. A general vapour-bath is administered in a wooden box-shaped apparatus, in which the patient sits entirely enclosed except for the head. The bath is usually administered at a temperature of 100° to 114°, for a period varying from 10 to 15 minutes. The local vapour-baths are administered in an ingenious apparatus, by which any part of the limbs can be submitted to the influence of the hot vapour. The temperature is often used as high as 117° for periods varying from 10 to 30 minutes. The local bath is often followed by douches, packs, and

possibly massage. This form of bath is frequently used in cases of acute gout, but especially in cases of old chronic joints, and in gouty affections of the skin.

The above are the baths most frequently used in gout, but it may be found expedient also sometimes to use baths with which is associated electricity in its various forms, or possibly the Nauheim system or the radiant-heat baths may be indicated. As these systems have all been fitted up in the Bath establishment, there is no difficulty in prescribing them in suitable cases, but as a general rule their use is not called for in the treatment of gout.

Combined with the mineral waters the patient's dietary has to be carefully supervised as well as the amount of exercise necessary for each case. Dry massage, electricity, and passive movements and exercise may also be indicated, and will call for careful consideration on the part of the physician.

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ARTHRITIS.

A COMMENT UPON AND REVIEW OF SOME RECENT LITERATURE UPON THE SUBJECT.

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INTRODUCTORY.

It is no easy task to grapple with the subject of arthritis even to the limited extent of stating in plain terms some of the current views on the subject and the evidence in their support, for around gout, rheumatism, and rheumatoid arthritis theory has cast her bright and attractive mantle, beneath which grey and sober fact is liable to be stifled. But there is little doubt that of recent years the principles of bacteriology have helped us forward a great deal in our knowledge of arthritis. They have brought us facts where there were once only theories, and they have opened up lines of investigation which eventually may bridge over the space between the humoral theories of auto-intoxication and those of intoxication or toxæmia from some bacterial infection.

It is strange how those who uphold the views of autointoxication—for example, uric-acid saturation—stand at daggers drawn with those who lean to bacteriological views. Yet when we think of gout and rheumatism, what is more likely than that through a study of bacterial toxines we may get a real insight into the processes of gout? If the poisons of the latter disease are due wholly or in part to a disturbed metabolism, these bacterial poisons are surely more akin to those due to a disturbed metabolism than is some crude chemical product such as uric acid.

Apart from these two divided camps, there are great difficulties of clinical interpretation. For some, bony outgrowths suffice to stamp a disease; for others, a drug such as salicylate of soda serves as the test. Others, again, claim a separation for two diseases, because in one case large and in another small joints are affected. What more bewildering chaos can be imagined, for example, than the affections grouped under the title rheumatoid arthritis? At present it would seem that the old methods of classification are being abandoned for a classification on bacteriological lines. We hear of pneumococcic, streptococcic, and gonococcic arthritis. But, unfortunately, the change is difficult to make, for bacteriology is a young science, and people ask for a sign where no sign can be given. They demand that some dye, some difference in the shape of a micrococcus, or some growth on a wholly foreign medium be made the test of a disease, and the result is vexation of spirit.

RHEUMATIC ARTHRITIS.

In considering rheumatic arthritis it is essential to recognise the view of the particular writer upon the subject. Does he mean by this a general term for the transient forms of inflammation, or, if chronic, for a stubborn inflammation not terminating in destructive suppuration? Or does he mean a form of arthritis which improves under the use of salicylates? Or does he mean a manifestation of a general constitutional disease, rheumatic fever? It is the last of these that I intend when speaking of rheumatic arthritis, and the Special number of THE PRACTITIONER for January, 1901, gives the first complete application of the view in this country, and can be used as a starting-point from which to date the more recent papers.

Since that time many papers have appeared on rheumatic fever[1] and rheumatic arthritis, in England and on the Continent, and it may be truly said that this conception of the disease has gained ground, and offers a clearer explanation of facts than any other one. It is in accord too with the clinical experience of the leading English clinicians. The view, stated in brief, is that a microörganism, variously described as a diplococcus, micrococcus, or streptococcus, is a cause of the disease. Whether it is the only bacterial cause is disputed; but, in my opinion, no one yet has demonstrated any other organism as a cause.

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[1] Among the recent papers the following may be mentioned:—

1. The discussion held by the Chelsea Clinical Society, published in the *Lancet*, *British Medical Journal*, and *Clinical Journal*, March, 1901.

2. Fritz Meyer: "Zur Bakteriologie des akuten Gelenkrheumatismus." *Deutsche Medicinische Woch.*, 1901, No. 1. In this Meyer published the isolation of a diplococcus from rheumatic angina faucium.

3. Poynton and Paine: "Further Investigaticns upon Rheumatic Fever." *Lancet*, May, 1901. This paper dealt with rheumatic chorea—the rheumatic nodule and the experimental production of Morbus cordis. Poynton and Paine: "Experimental Rheumatic Arthritis." *Path. Soc. Trans.*, June, 1901.

4. Singer: "Ueber die Aetiologie des acuten Gelenkrheumatismus." *Wiener Klinische Wochenschrift*, 1901, No. 20. In this the author maintained that the diplococcus of Meyer was the attenuated Streptococcus pyogenes, and that more than one microörganism might be a cause of articular rheumatism.

5. Allaria (*Rev. Crit. de Clin. Méd.*, November 23, 1901) confirmed the observations of Poynton, Paine, and Meyer upon the relation of angina faucium and arthritis.

In 1902, Menzer published his interesting lectures "Ueber Angina, Gelenkrheumatismus, Erythema nodosum, und Pneumonie, nebst Bemerkungen über die Ætiologie von Infectionskrankheiten" (*Berl. Klinische Wochenschrift*, 1902, 1 and 2), and shortly afterwards his monograph upon "The Ætiology Acute Articular Rheumatism" (Berlin, 1902).

In April, 1902, Poynton and Paine[1] demonstrated that the micrococcus of rheumatic fever might produce malignant endocarditis, and suggested that this might throw light on rheumatoid arthritis in rheumatic subjects.

In the same year, Fritz Meyer[2] published an extensive paper upon the bacteriology of acute rheumatic fever arthritis. In this paper he contributes many interesting points to the scientific study of acute articular rheumatism. He gives important and independent confirmation of the facts put forward in earlier papers; that is to say, he found the diplococcus or micrococcus in more cases of rheumatic tonsilitis, and by inoculation with it produced arthritis, endocarditis, and pericarditis. In three rabbits out of 100, he noted peculiar twitching movements, which he believes may be an expression of chorea. Writing of the arthritis, he points out its synovial origin, and dwells on the rapid destruction of the bacteria by cellular activity. He believes that the micrococcus has a remarkable affinity for the joints, and that if staphylococci and

6. Predtetchensky (*Vratch*, No. 24, p. 761, 1901) brought forward more confirmatory evidence upon the micrococcus, and produced the symptoms of rheumatic fever in guinea-pigs.

7. Brill and Libman (*Academy of Medicine*, New York, May, 1901) again brought forward confirmation.

[1] "A Contribution to the Study of Malignant Endocarditis," *Trans. Royal Med. Chir. Soc.*, Vol. LXXXV.

[2] "Zur Bakteriologie des acuten Gelenkrheumatismus," *Zeitschrift für Klin. Medicin*, 46 Bd., H. 5 and 6.

diplococci are injected, the diplococci will be found in joints before the staphylococci. This statement he supports with experimental data. His experiments, conducted on a large scale, have also led him to believe that the diplococcus has a special affinity for the cardiac valves. 20 per cent. of animals injected developed endocarditis. This paper brings into especial prominence the importance of tonsillar inflammations in the causation of rheumatic arthritis.

In 1903, Ainley Walker[1] brought forward important confirmation of previous investigations as a result of a research with Beaton. In addition, he pointed out that the micrococcus, as he terms it, can grow in a filtered culture of the Streptococcus pyogenes. This, following the lines of Marmorek's[2] investigation, published in 1902, is the first effort in this country to differentiate the microörganism by tests other than experimental.

I fear that this enumeration of writings must be tedious, but it will, I think, assist those who are especially intent on following the drift of recent work. The papers alluded to are mostly technical ones dealing with the pathology of rheumatic fever, and with rheumatic arthritis.

It will also serve a useful purpose to mention the recent criticism of Triboulet[3] upon them, for it is impossible to keep upon this subject that freedom from bias which is so essential in presenting a review of recent investigations. Triboulet maintains that there is a simple articular rheumatism, non-microbic, or at least not due to the micrococcus; and that the micrococcus or diplococcus itself is the cause of the complications. He holds that there are numerous cases of polyarticular rheumatism, which are sterile on bacterial investigation, in which no complications arise. This he looks upon as the genuine rheumatism.

It would not be in accord with the intention of this brief review to leave the impression that this question of the pathology of rheumatic fever and rheumatic arthritis was settled. There are writers of experience, acquainted with the bacteriological work on the subject, who maintain that the cause must

[1] THE PRACTITIONER, February, 1903, p. 185.

[2] *Annals of the Pasteur Institute*, January, 1902.

[3] Triboulet: "Le Diplostreptocoque dans le Rhumatisme articulaire aigu," Étude Critique, *Gazette des Hôpitaux*, April 4, 1903.

be sought elsewhere. The late Dr. Maclagan, whose name will always be remembered with respect and gratitude, held to the end that the infection was more akin to malaria. Dr. A. Haig's views are too well known to need mention here.

Dr. Heinrich Stern, in an elaborate paper,[1] maintains that the rheumatic process is a result of an increased permeability of white fibrous tissue, particularly that of the serous and synovial membranes. This increased permeability is effected by the calcium-salts of the blood, which extract the cementing substance from the fibrous texture. Dr. Stern admits that there is a certain amount of hypothesis in the theory, but far less than in the bacterial and lactic-acid theories, and according to him acute rheumatism is rare in childhood.

The most definite evidence against the micrococcal explanation of rheumatic fever and arthritis is that published by Thomas McCrae.[2] This is an interesting analysis of 270 cases from the Johns Hopkins Hospital, and the author remarks that the bacteriological results were practically negative as to urine, blood, and arthritic exudation.

In concluding this section it may be pointed out that the bacterial view of rheumatic fever and rheumatic arthritis is a question of the interpretation of observed facts rather than a matter of theory.

RHEUMATOID ARTHRITIS.

It is most difficult to arrange the writings upon rheumatoid arthritis, and I think it must be considered a matter of real scientific and practical importance for the medical profession to agree, whether by this term is to be meant a special disease or a number of intractable joint-affections.

If it is a special disease, let its limitations be laid down in such a way that investigators can recognise that they are studying the same disease; if it is only a term for any intractable joint-affection, then it is to be expected that it owns many causes, and we shall be prepared to find that many different results will be obtained by these investigators.

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[1] "Acute Articular Rheumatism," *Medical News*, New York, August 23, 1902.

[2] "Acute Articular Rheumatism," *The Journal of the American Medical Association*, January 3, 1903.

Discussions upon the subject of rheumatoid arthritis have been frequent of late in this country, for example :—

1. The discussion at the Chelsea Clinical Society in March, 1901.

2. At the Cheltenham meeting of the British Medical Association in August, 1901.

3. At the Manchester Medical Society in January, 1903.

In January, 1902, Poynton and Paine,[1] isolated a diplococcus from a case of rheumatoid arthritis in a man aged 67, who had died by misadventure from carbolic-acid poisoning. By intravenous inoculation of rabbits, they produced not only a severe multiple arthritis, but also an osteo-arthritis of the monarticular type. This investigation settled once and for all that a microörganism obtained from a joint which is the seat of osteo-arthritis is capable on intravenous inoculation of producing the lesions of rheumatoid arthritis; also that a monarthritis may result from an infective agent, not introduced directly into the joint. We can now say that an infective agent introduced into the blood has the *power to produce* osteo-arthritis, whereas before we were only justified in saying that an infective agent is frequently *present* in osteo-arthritis.

In March, 1902, Page, Poynton, and Paine published "Two Cases of Arthritis of the Knee."[2] In one case during life, diplococci not to be distinguished from those found in rheumatic fever were demonstrated in, and cultivated from, the exudation in the knee-joint. Both rheumatic and rheumatoid changes were produced by the microörganism injected intravenously into rabbits. This result, coupled with the results obtained with simple and malignant endocarditis, points strongly to the probability that some cases of rheumatic arthritis may develop the rheumatoid character without necessarily the addition of a secondary infection.

In 1902 Hale White[3] described in detail two cases of acute rheumatoid arthritis, one of which died from some cause not clearly determined. In this case pathological investigations were made by Fawcett and Stevens. The bacteriological investigations were made in the laboratory at Guy's Hospital

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[1] *Path. Soc. Trans.*, Vol. I., 1902.

[2] *Clin. Soc. Trans.*, Vol. XXXV.

[3] *Guy's Hospital Reports*, Vol. LVII. **Hale White: "On the Pathology of Acute Rheumatoid Arthritis."**

(the name of bacteriologist is not mentioned), and the skiagraphy of the joints was done by Shenton. Hale White is in agreement with Bannatyne and A. E. Garrod, that this acute form should be separated from the chronic. He is of opinion that it is a condition quite distinct from that of rheumatic fever, and relies for this upon the previous history, the age and distribution of the lesions, together with the absence of other manifestations such as chorea, pericarditis, &c.

The pathological changes in the bones and joints were those of inflammation, the synovial membranes especially being affected, and to some extent also the periarticular tissues. There was no proliferation of bone or cartilage. There are interesting photographs of the joints with remarks upon them. The exceptional transparency of the bones, first pointed out by Walsh in 1897, is well seen in them, and the importance of this condition is emphasised by the writer. (In passing it may be added that Triboulet in 1901 pointed out a still earlier change close to the epiphysial cartilage of the bones of the damaged articulations, which is detected by the Röntgen rays.)

The bacteriological results in Dr. Hale White's case were as follows :—From a mesenteric gland a small non-motile bacillus, non-pathogenic to rabbits or mice, was isolated. This organism stained by Gram's method. From the left knee-joint a diplococcus, which was non-pathogenic, was also isolated. The author remarks that it was apparently not similar to that described by Poynton and Paine, nor did it resemble the more usual cocci.

Bannatyne, whose pioneer work in this country on the infective nature of rheumatoid arthritis is so well known, has in "Notes on Rheumatoid Arthritis" put together in a pamphlet some interesting papers on the subject of arthritis. These papers deal with the following :—

(1) Points in the classification and diagnosis of some joint-affections.

(2) The relationship between acute infective rheumatism and rheumatoid arthritis.

(3) The anæmia of rheumatoid arthritis.

(4) The pathology and treatment of the disease.

(5) Excision of the joints in some cases of arthritis.

Gask[1] isolated a streptococcus from a case of acute rheumatoid arthritis in a woman aged 29 following delivery. Both in films and on culture were demonstrated streptococci indistinguishable from the Streptococcus pyogenes. The organism proved non-virulent to a mouse and a rabbit.

Writing upon this question of the bacteriology of rheumatoid arthritis, Stengel[2] remarks: "Certain investigators, notably in England, have claimed the discovery of microörganisms in rheumatoid arthritis. These claims, however, do not merit serious consideration." This is naturally a matter of opinion, but for the words "claimed the discovery" may fairly be substituted "shown the presence of."

Jones[3] describes cases of the spindle-type of arthritis following upon the symptoms of Raynaud's disease, and believes that the phenomena of the articular and periarticular swelling are in a similar manner dependent upon vasomotor spasm. He also describes transient obscurations of vision in rheumatoid subjects, dependent, as shown by Beaumont and himself, upon a contraction of the visual fields. This symptom was only present in those cases of rheumatoid arthritis in which the symptoms of Raynaud's disease were well marked, and it showed some correlation with the affection of the temporo-maxillary joint. In addition ptosis of the upper eyelid may occur, and even paralysis of the external ocular muscles. Beaumont noted excessive hippus. Jones points out an association between Graves' disease and rheumatoid arthritis. He believes that these symptoms point to a poison in the blood acting on the cerebro-spinal system through the medium of the blood-vessels, which are provoked by this poison to spasmodic contraction.

Macalister[4] lays stress on the importance of a nasal infection in some obstinate cases of arthritis. The paper is based on a study of six cases of arthritis, and the writer looks upon the action of salicylates as a test of the rheumatic process.

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[1] *Lancet*, May 9, 1903.

[2] "Articular Rheumatism and some Allied Conditions," *The American Journal of the Medical Sciences*, March.

[3] R. Llewelyn Jones: "Rheumatoid Arthritis as a Cerebro-spinal Toxæmia," *Edinburgh Med. Journal*, January, 1903.

[4] "Some Diseases simulating Acute Rheumatism, and their probable relation to Chronic Arthritis," *Medical Press*, December, 1902.

TUBERCULAR ARTHRITIS.

In a recent paper[1] Poncet expands his earlier writings upon the subject of "Le rhumatisme tuberculeux articulaire, ou pseudo-rhumatisme infectieux d'origine bacillaire." Unfortunately the term rheumatism is used here in a different way to that in which we, as a rule, use it in England. It is used here as a synonym for a transient arthritis. The type of arthritis of which he writes is then an acute multiple tubercular arthritis. As he had earlier pointed out, the usual chronic tubercular arthritis is but one phase—a very important one, it is true—of tubercular arthritis, and in addition there is this transient arthritis. In this paper he shows that not only the joints, but also the other serous membranes, may show the same transient inflammation. The morbid anatomy of such inflammation resembles that of other diseases, but experiment proves, by the development of tuberculosis in guinea-pigs, their tubercular nature. The same author (*Gazette des Hôpitaux*, January 20, 1903) describes in detail a case of tubercular pseudo-rheumatism.

Rolleston[2] describes a case of general miliary tuberculosis with acute synovitis. After death no tubercle was seen in the joints, but the other organs were riddled with miliary tubercle. One such case I have also met with in which only the knee-joint was thus affected, and the diagnosis from the severity of the symptoms hovered between osteo-myelitis with effusion, typhoid fever, and tuberculosis. Tubercular meningitis developed a few days afterwards, and the case proved rapidly fatal. As Rolleston points out, the condition is extremely puzzling, and rheumatism is usually diagnosed. The Lyons School of Medicine has done much to elucidate this subject.

J. Hobbs[3] calls attention to monarticular arthritis of the rheumatic type in the tuberculous.

PNEUMOCOCCIC ARTHRITIS.

No form of arthritis gives a better illustration of the gradual change that is taking place in our views on arthritis and bacteriology than this one. The pneumococcus was formerly looked upon only as a cause of lobar pneumonia, a disease essentially non-suppurative, but now we accept as rational the view that the same organism may be the cause of an arthritis which is often suppurative. Cave,[1] in an important paper, drew attention to this condition which had been carefully studied by Leroux[2] in France. Nathan Raw and others since that date have also added to the literature of the subject.

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[1] Poncet: "Rhumatisme tuberculeux articulaire," *Lyon Médicale*, July 20, 1902.

[2] *Lancet*, January 17, 1903.

[3] *La Presse Médicale*, February, 1903.

Cave, writing of the clinical characters, points out their variability, but distinct tendency to develop into a severe form of suppurative arthritis. The prognosis is therefore grave. An important diagnostic point is the association of this form of arthritis with pneumonia or with other local evidences of pneumococcic infection. Cave tabulates 31 cases—30 of them recorded by continental observers, in 28 of which pneumonia occurred. It is an interesting point that the arthritis usually supervenes after the pneumonia has run its course.

The diagnosis, when there is no evidence of pneumonia, will rest upon the bacteriological demonstration of the pneumococcus in the exudation, and a study of its characters. This microörganism, when contrasted with the micrococcus of rheumatic fever, will be found larger, more lanceolate, capsulated, more strictly a diplococcus, and staining more readily by Gram's method. It is more delicate in its growth, especially in acid media, and usually on experimental inoculation with rabbits produces a rapid septicæmia, and capsulated diplococci are found in the blood.

Howard Marsh[3] devoted his Bradshaw lecture to the question of infective arthritis, and he dealt in this with pneumococcic arthritis, typhoid arthritis, proper and septic arthritis in typhoid, scarlatinal arthritis, arthritis in erysipelas, unclassified cases of arthritis, and the acute arthritis of infants. He also alluded to arthritis in influenza, and to this observation I will refer again.

The lecturer pointed out the importance of the infective agent, and dwelt upon the importance of treatment. He grouped the cases into four types: (1) The acute and transient requiring little more than rest; (2) The second group requiring irrigation either with or without free opening of the joint; (3) The plastic form in which much care and judgment is needed in undertaking active interference, and in which one must often be content with imperfect results; and (4) Lastly, cases in which arthritis is only a feature in a general and usually fatal septicæmia. This contribution to the study of arthritis based on a wide experience and mature judgment is one of great practical importance.

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[1] "Pneumococcic Arthritis," *The Lancet*, January 12, 1901.

[2] *Les Arthrites à pneumocoques*," J. Rousset, 1899.

[3] "Infective Arthritis," *Lancet*, December 13, 1902.

Poynton and Paine,[1] in a further paper, deal with the diagnostic value of bacteriology in infective arthritis, and add some remarks upon its limitations. The authors lay especial stress upon the value of an accurate description of the exudation and the avoidance of the term "septic." This, as now used, conveys but little, if any, accurate idea of the nature of the infection. Examples are given of various forms of arthritis which they had investigated.

INFLUENZAL ARTHRITIS.

Influenza has been followed in some cases by a very troublesome arthritis. Rheumatic patients and those suffering from rheumatoid arthritis have been repeatedly noticed to have severe outbreaks after influenza, but in addition there are some instances in which arthritis seems to be directly due to the influenzal infection. Such cases are intractable, and may be accompanied by much swelling around the joints, and implication of the tendon-sheaths. It is a painful and crippling affection.

Howard Marsh[2] describes two such cases in which the hip-joints were affected, and points out the close relation of the arthritis to an epidemic of influenza, the slow course, the œdema, and the pain and muscular wasting.

F. C. Wallis[3] also gives an account of arthritis following influenza.

The cases are of much scientific interest, because it is generally recognised that influenza is an infection and also a powerful nerve-poison. We have then, when arthritis occurs, that combination of nervous disorder and arthritis which is so emphasised by many writers upon rheumatoid disease. Yet no one would claim the arthritis in these influenzal cases to be the direct result of the nerve-disturbances, but rather a manifestation of the infection. Much the same process of reasoning is probably to be applied to genuine rheumatoid arthritis.

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[1] "Observations upon Certain Forms of Arthritis," *British Medical Journal*, November 1, 1902.

[2] *Op. cit.*

[3] "The Diagnosis and Treatment of various forms of Septic Synovitis" *Lancet*, January 3, 1903.

GOUT AND GOUTY ARTHRITIS.

This difficult subject is perhaps best thought of at present as in a stage of critical enquiry rather than of visible advance. The great stimulus given to the subject by Sir Alfred Garrod —who is happily still among us—has led to a vast amount of investigation, some of which has fallen into a too narrow groove, with the result that in some quarters for the clinical condition "gout" there has been substituted, as an equivalent, the chemical body "uric acid." Side by side with this we trace a revulsion of opinion which, so far as it rests upon a basis of fact, must throw light upon the disease.

Of this revolutionary type is the paper by Woods-Hutchinson.[1] Among his conclusions are the following :—

1. That between the production of urea and uric acid there is no connection, and that therefore marked limitation of animal or nitrogenous foods, as such, is irrational.

2. That of the two sources of uric-acid formation in the human body, exogenous and endogenous, the latter, due to the destructive metabolism of the nucleïns in the body-tissues, is alone increased in gout and lithæmia.

3. Gout is merely a name for certain processes, miscellaneous in origin, which are characterised by the production of uric acid and urates.

4. The gouty diathesis is the expression of an inadequate resisting-power of the body to oppose the entrance of any poison, whatever its character or source.

5. That uric acid, equally with phosphoric acid, is an index, as it were, of the degree of damage done by such poisons;

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[1] "The Meaning of Uric Acid and the Urates," *Lancet*, January 31, 1903.

hence the action of solvents is irrational, since these products are not the essence of the disease.

6. That diet in gout should be regulated solely with a view to the diminution of intestinal fermentation and putrefaction.

7. That the limitation of animal foods is indicated, because these are the most appetising and the most liable to be indulged in to excess.

8. Since uric acid is not toxic or only feebly so, it is not rational to prohibit foods rich in nucleïns or similar bodies, except in so far as they tend to increase the attractiveness of the diet and thus lead to overfeeding.

In this paper there are comments on the various theories and some statements upon treatment to which space prohibits allusion.

Poynton and Paine[1] called attention to the acid-producing properties of the micrococcal infections, especially that of rheumatic fever. Milk when made distinctly alkaline is rapidly acidified and clotted. Ainley Walker[2] also commented upon the acid-producing properties of this organism and its possible bearing upon gout. It would seem very possible, seeing the close relationship between gout and rheumatism, that an acute gouty attack (not gout) may be precipitated by such an infective agent.

THE TREATMENT OF ARTHRITIS.

The treatment of arthritis must necessarily advance slowly and with caution, but recently we recognise the development of two lines of procedure. One of these, it is true, is yet in its earliest infancy, but the other has been the subject of several recent papers. The former is the treatment by some protective serum, the latter that of active surgical interference. Both are an outcome of the modern views on infective diseases. So long as we were tied and bound by mysterious diatheses in which the sufferer brewed, as it were, within himself a continual supply of disease, it was a difficult matter to get even reasonable consideration for such treatment, but it seems very probable that in the future we may learn much of value in these directions.

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[1] "A Contribution to the Study of Malignant Endocarditis," *R. Medico-Chir. Soc. Trans.*, 1902.

[2] THE PRACTITIONER, February, 1903.

SERUM-THERAPEUTICS.

So important is the question of serum-therapeutics that it may be useful to dwell briefly upon some of the difficulties that have to be faced by those who undertake this method in the laboratory, or—what is of more importance—its practical application in disease.

First and foremost these antistreptococcic sera are, as a rule, in principle, antibacterial; that is to say, they are intended when injected to destroy the bacteria which have gained access to the tissues, and they are not antitoxic in the sense that the diphtherial serum is antitoxic. This difference is one of cardinal importance, for it is notoriously more difficult to obtain success with a serum of this kind.

There are often enough great difficulties in obtaining such sera, because in the streptococcal group there are micro-organisms which it is extremely difficult to maintain in a virulent condition, and with which it is, therefore, difficult to obtain a lethal dose for animals. It is equally difficult also to raise their virulence. The usual principle is to raise this virulence by passing the organism through a series of small animals, and Aronson has claimed success in some of the more difficult by passing them through white mice. When the virulence has thus been raised larger animals are used, and in this way a sufficient quantity of serum obtained.

There are also practical difficulties in making sure that the serum is not itself toxic, and I would refer the reader to Professor A. E. Wright's lecture on the "Therapeutic Inoculations of Bacterial Vaccines"[1] for a more complete account of this question, with the description of the immunity-curves in their negative and positive phases.

When dealing with antibacterial sera, it is necessary to remember that because a serum immunises an animal, for example, a mouse, it does not follow that it will immunise a man. Further, the antibacterial elements in the serum are apparently unstable and rapidly disappear. This fact Professor Wright points out in the same paper.

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[1] *Brit. Med. Journ.*, May 9, 1903.

So utterly obscure is the chemistry of these processes that it is quite open to speculation whether the mere passage through the animals may not alter the therapeutic action for man. For this reason Tavel and Krumbein[1] have insisted upon the importance of avoiding the use of small animals. They have taken cultures direct from man, and by using large quantities of these have produced—without the intervention of small animals—results in large ones, such as horses.

Again, there are difficulties concerned with the application of these sera. It is said, often enough, that such and such a serum can do no harm, but personally I accept this statement with great caution. This is certain, that some observers, for example Baginsky,[2] have made very definite statements as to serious effects of this method of treatment, and assert that they produce very definite reactions, which will be evident from Menzer's results shortly to be mentioned. Enlargements of the liver and spleen, rigors, joint-swellings, rashes, general malaise and pains have all been noticed. Nor is the continual hypodermic injection of fluid under the skin altogether free from objection.

Lastly, great care is necessary on the part of the physician in judging of the therapeutic results. If a serum is antibacterial, it would seem logical to push it at first in considerable quantities, while there is evidence that the disease and probably therefore the bacteria are especially active. Yet often enough cases are reported in which this is not done. Again, in cases in which there is fever of the septic type, the temperature rising each evening several degrees and falling just as rapidly, serum is injected at the height of the fever and the subsequent fall attributed to its use, when a more certain test would be to inject it just as the temperature commences to rise, and then to ascertain if it controls the fever. The tendency of rheumatic fever to abate of itself, sometimes abruptly, adds to the difficulties of the physician.

These remarks may, perhaps, show what a difficult question is this one of serum-therapeutics, and how easily we may be led astray, and even make a retrograde step in medical treatment, unless each point is carefully studied.

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[1] "Ueber Streptocokkenserum Therapie," *Correspondenzblatt für Schweizer Aerzte*, No. 8, 1901.

[2] *Berliner Klin. Woch.*, December 1 and 8, 1902.

MENZER'S ANTIRHEUMATIC SERUM.

Reference, in some detail, will now be made to Menzer's paper[1] on the serum-treatment of acute and chronic rheumatism.

Menzer obtained his cultures from the upper air-passages of his patients suffering from rheumatic angina, and thus obtained streptococci often in pure culture. The method of manufacture of his serum he does not detail, but states that it was prepared for him on Tavel's principle by the firm of E. Merck under the supervision of Dr. Landmann. The point of view which he takes as to the bacterial cause of rheumatism is that it is not the result of a specific micrococcus, but is nevertheless usually due to streptococci. The peculiar symptoms of acute rheumatism are ascribed by him as mainly due to the *resistance* of the human tissues to these streptococci.

His serum produces certain very definite effects in subacute cases. After four to six hours, redness, swelling, and heat occurred in the joints which had been affected by the rheumatism—in these and in no others. For a while also the cardiac symptoms appear to have been augmented; there were fever and pains in the neck and head, swelling of lymphatic glands, and sore-throat. Later there was a fall of the temperature by lysis, and then cure.

He gives at length seven cases of acute rheumatism treated by this method. It is difficult from a mere perusal of the cases to feel convinced that they benefited, though none of them had relapsed at the time of writing. A case of rheumatism with nodules made a rapid recovery. A case of rheumatism with nephritis improved, but each injection of serum was followed by a slight exacerbation of the nephritis.

With chronic rheumatism he claims better results and cites some remarkable cases. The first result of the injections was to light up the arthritis and to make it for the time acute; there then followed recovery.

He next gives examples of septic conditions which benefited by this treatment and showed reactions similar to the rheumatic cases—a point which confirms his view of the non-specific nature of the rheumatic lesions. He further suggests that the serum may be useful for the treatment of chronic nephritis, for though it has no action on the healthy kidney, it causes a local reaction in a damaged kidney evidenced by the appearance of blood and casts.

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[1] Menzer: "Serum Behandlung bei acutem und chronischem Gelenk-Rheumatismus," *Zeitschrift für Klinische Medicin*, Berlin, 1902.

He does not claim that the serum cures rheumatism in the ordinary sense of the word, nor that large doses will abort rheumatic arthritis, but that it allows the natural resistance, which is failing or has failed, to reässert itself, and the disease then takes a natural course towards cure.

As yet no test of the protective power of the serum has been obtained, and the correct dose must be gauged by cautious trial. His test-dose is 5—10 c.c., but as much as 200 c.c. have been used for a single case. He believes that it prevents relapses and the development of endocarditis, but in cases with very high fever or with much pericarditis or pleurisy it requires caution in its use because of the local reactions.

In the discussion which followed the reading of the paper, Blumenthal compared the action of this serum to Koch's tuberculin, for he remarked that if it were antibacterial or antitoxic in action the flaring up of the disease (*i.e.*, the local reactions) would not have occurred. He was of opinion that Menzer had produced an active immunisation, to do which he need not have used animals at all. Menzer in reply denied this, stating that all sera made by Tavel's process were antibacterial.

The use of a serum which produces such local effects as this of Menzer's must naturally cause anxiety; for if there is no accurate method of testing its power, we should dread that its action should go further than was expected, and do harm rather than good. The completeness of this paper and the skill with which it is written makes it one of the most remarkable of recent writings upon rheumatism.

I have for some months, under close observation, tried an antistreptococcic serum in carefully chosen cases of rheumatic pericarditis, endocarditis, arthritis, and chorea. In my opinion it was quite useless either in cutting short the disease or preventing relapses. It was manufactured from various strains of streptococci and obtained from the Pasteur Institute.

SURGICAL TREATMENT.

The surgical treatment of arthritis other than true suppurative arthritis has of late come a good deal to the fore, and there appears some hope that in suitable cases we may do material good. I would refer the reader again to Howard Marsh's Bradshaw lecture for information upon this point. The writer very clearly points out that there are a considerable number of intractable cases in which cautious surgical interference will do good, though often no very brilliant result is to be expected.

Mr. Page[1] quotes two cases of stubborn arthritis of the knee treated by lavage with 1-per-cent. carbolic-acid solution; and Mr. F. C. Wallis[2] also gives examples of cases of arthritis benefited by surgical treatment.

J. O'Conor[3] writes a most remarkable paper on the surgical treatment of acute rheumatic arthritis. In brief, he advocates arthrotomy for acute rheumatic arthritis, and supports his contention by details of twenty cases thus treated. He claims that this method leads to an early cessation of the general toxæmia and protects the heart against damage. It is with the acute condition of arthritis that he has obtained his greatest successes, and his results are apparently good.

Whenever some striking alteration in a method of treatment arises, it is needful to question closely the why and wherefore. We wish there had been bacteriological proof of the nature of his cases before we pledge ourselves to such radical treatment. Again, are we in a position to state that the cure of rheumatic arthritis by surgical measures will arrest or prevent the cardiac infection? This is a very important point. So far as one can see at present the infection in rheumatic fever appears to be a general one. The heart and joints for example seem to suffer from the infection directly, rather than the one to arise as a result of the other. This view of a general infection by no means implies that all the lesions should appear clinically at the same moment of time. Lastly, the free use of the knife is not a method that physicians and patients take to kindly, since acute rheumatic arthritis does so well without such treatment. These thoughts will doubtless occur to all who read the paper, but there is no doubt that O'Conor has made important statements which he has supported by definite and clearly-stated facts.

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[1] *Clin. Soc. Trans.*, 1902.

[2] *Lancet*, January 3, 1903.

[3] *Lancet*, January 24, 1903.

TREATMENT BY DRUGS.

The treatment of arthritis by drugs has made no great advance, but further experiences of the use of aspirin have led to the opinion that it is a real addition to our remedies. Syers[1] considers the drug superior to salicylate of soda for the relief of pain, whether it be the result of acute or of chronic rheumatism, and has found it reduce the temperature when salicylate of soda has failed. It may cause vomiting in children—a fact which I mention in passing, because the impression is sometimes given that it never produces any ill effects.

ARTHRITIS DUE TO INJECTIONS OF SERUM.

In conclusion, some facts are gradually accumulating on the production of arthritis by the experimental injection of sera. In Menzer's investigations arthritis recommenced as a result of the injections. Arthritis has been observed from Koch's tuberculin. Pains in the joints without swelling have been repeatedly observed after treatment with anti-diphtheric serum. These are very interesting points, for though we know much of arthritis as a result of bacterial invasion of the joints, we know little of any poisons produced by bacteria, which can, without the presence of these bacteria themselves, produce arthritis. Research in these directions may throw upon gout and rheumatism and rheumatoid arthritis, especially upon the first, much valuable light.

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[1] *Medical Times and Hospital Gazette*, November 15, 1902.

Notes by the Way.

Gout.

THE phenomena of an attack of gout are so well marked as to constitute an easily-recognised disease. Thus it has been known from the earliest times. Hippocrates was well acquainted with it, and references to it abound in classical authors. The chief theme of Lucian's well-known Dialogue is the torments caused by the disease and the uselessness of remedies: time and endurance are the only helpers against this Avenging Deity. Even at the present day we cannot claim to know much beyond the clinical features of the malady. Its true causation is still very far from being ascertained, and the problems underlying its various manifestations still await solution. In our present number we have endeavoured to gather together the views of the chief authorities on the disease in this country, and our best thanks are due to our contributors for the valuable articles in which they have discussed the different sides of the subject. On a matter so much debated it was not to be expected that the views of all would coincide; yet perhaps a study of the different articles, and an application of the "method of agreement and difference," as the logicians would say, reveals more community of ideas than might have been expected. In the mind of the average student of the subject the idea of gout may perhaps be summed up in the expression "uric-acid poisoning," and we find that in Dr. Haig this view of the matter has a redoubtable champion. Indeed, this writer goes so far as to hold that, not only gout, but also rheumatism and rheumatoid arthritis, are due to this peccant matter. In this extreme view we do not think that he will find many supporters, as the tendency of opinion at the present day seems to be more and more in the direction of regarding acute rheumatism as a microbial disease. A very clear account of recent investigations in this field was given by Dr. Ainley Walker in the February number of THE PRACTITIONER for this year. We confess that we are not prepared, on the evidence hitherto available, to identify the disease with gout as to nature and causation. In the latter, however, there is as yet no reason to suspect a bacterial cause; indeed, everything points to a failure in the chemical processes of the body as the underlying factor in this malady. The fact that it is associated with the presence of an excess of urates in the blood and tissues has been fairly established by the classical experiment of Sir William Roberts, who showed that by acidulating the blood in a gouty patient it was possible to demonstrate crystals of uric acid deposited on a thread suspended in the fluid. This demonstration does not necessarily prove that the excess of urates is the cause of the disease. It would be equally possible to regard these substances as effects rather than causes of the condition. Even in the joints where the deposits of urate of sodium are so well known and so characteristic, it does not necessarily follow that the salt is the excitant of the accompanying inflammation. Apart from definite proof, it is as feasible to hold that the urate is thrown out because the joint is diseased, as that the joint becomes diseased because the urate is deposited there.

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Uric Acid as a Poison.

THAT uric acid is a poison our contributors seem agreed. Dr. Haig refers to the possibility of demonstrating this by experiments on oneself, but advises caution in so doing or the effects may be disastrous. Dr. Walker Hall also regards the substance as toxic, showing that it acts as an irritant on mucous membranes. It is necessary to bear in mind that in the body it is not actual uric acid which is circulating in the blood and getting deposited in the tissues, but urate of sodium. It is more important to know if this latter is seriously poisonous. But it is not only to uric acid itself that attention has been directed. Many allied bodies exist belonging to the so-called "alloxur" group, to which the name of "purins" has recently been applied. Modern chemistry has made these substances the subjects of careful study, and their importance in the pathology of gout is probably considerable. Our readers will find a very exhaustive account of the formation of these bodies and their relation to gout in Dr. Walker Hall's article.

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Origin of Uric Acid and its Allies.

It seems that uric acid and the purin-group of bodies are derived from the "nucleins" or materials of which the cell-nuclei consist. The breaking-up of these nuclei gives rise to uric acid and the alloxur-bodies. Now it is clear that two possible origins of these substances as met with in the body are thus conceivable, viz., from the nuclei of the cells of the living body itself and from those of the animal and vegetable matter consumed as food. Both of these may give rise to uric acid in the system, the food-nuclei causing what is known as the exogenous, the body-cells the endogenous, supply. An excessive supply of nuclear products may thus arise from the consumption of kinds of food rich in these elements, or from some perverted metabolism of the body-cells. To which of these is the condition known as gout due? We cannot, perhaps, as yet answer the question, though Dr. Haig strongly holds that gout is due to food-poisoning, and to that alone.

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Gout and Renal Disease.

But it may be asked, is it certain that there is an increased formation of the substances to which we have just alluded? Is it not equally possible that it is rather a failure to get rid, by excretion, of the normal supply of them which leads to their appearance in excess? The frequent association of gout with renal inadequacy in the form of the granular contracted kidney renders the solution of this question difficult, while adding to its importance. Is the disease of the kidney the primary lesion in these cases, or does the poison which gives rise to the gout damage the kidney and cause its degeneration and fibrosis? Probably the latter is the case, as instances of gout may be met with presenting no ascertainable lesion of the kidney. But it is not impossible that the disease may arise in two ways, either by increased production of the poisonous material or by its defective elimination. Still the close connection between gout and such disorders as diabetes and obesity seems to point to its origin in a perversion of the general metabolism of the tissues; but in what this consists, or what brings it about, is practically unknown.

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Causation of Gout.

CERTAIN factors in its causation are, however, pretty accurately determined. Thus, of the hereditary nature of the malady there is room for no doubt whatever. Probably this fact has much to do with the incidence of gout on particular races, our own country being the greatest sufferer from gout, whereas on the Continent of Europe it is comparatively rare. It is more common among the rich than the poor, and has been called "morbus divitum" in consequence. Over-feeding and luxurious habits of life undoubtedly favour its onset; yet it may occur in the most abstemious at times, and the poor are not exempt from its attentions. It might seem that the poor who suffer from gout would afford more favourable subjects in whom to study its causation than the rich, on account of the less varied nature of their dietary and conditions of life. The article by Dr. Toogood in our present number points strongly to one, and only one, source of the disease among the labouring classes—malt-liquors In view of this evidence, it can hardly be doubted that this class of beverages constitutes at least a potent exciting cause of gout. The comparative infrequency of indulgence in malt-liquors by women may account for the smaller incidence of gout on this sex, while the prevalence of the disorder in England as opposed to the Continent may also be due to the nature of our prevailing beverage. Yet in predisposed individuals gout may almost certainly arise in the absence of this causal factor.

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Gout and Lead.

ANOTHER well-known cause of gout is lead-poisoning. Not only do painters and workers in lead tend to suffer from gout, but the administration of a dose of a lead-salt, such as the subacetate, to a gouty patient may bring on an acute paroxysm. What is the connection between cause and effect here? Again we have to confess that we do not know. It is true that lead has an ill effect on the kidneys, chronic poisoning with this metal being accompanied by the changes in the kidneys which we know as chronic interstitial nephritis or granular kidney. Perhaps the ingestion of a single dose impairs the secretory function temporarily, and so brings about some retention of urates. But it is at least equally possible that it leads to some sudden precipitation of these salts, or produces some unknown change in the chemical functions of the tissues, which is the cause of a gouty attack.

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Gout as an Auto-intoxication.

A PAPER appeared not long ago, written by Dr. Woods Hutchinson,[1] in which the causation of gout was attributed to the circulation in the system of poisons formed in the alimentary canal as the result of abnormal fermentation or decomposition of food therein. We know little of such intestinal and gastric poisons. They may theoretically be formed either by the action of unusual bacterial inhabitants of the alimentary canal, in which case the condition is practically one of infection, or by perversion of the normal gastric, pancreatic and intestinal juices. The best-known instance of such poisoning is that of tetany, in which toxic products are formed in a dilated stomach. Other diseases, as for instance many forms of eczema, are very probably closely connected with conditions prevailing in the digestive tract. That such may be the case with gout is very far from impossible. Perhaps such defects of digestion are the starting-point of chemical changes affecting the assimilation of the nuclear substances to which we have already alluded.

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Goutiness.

A FRANK attack of gout is easily recognised, but how often are patients described as gouty who have never had a true paroxysm! It is difficult to feel sure whether we are justified in speaking of goutiness apart from actual gout. Certain it is that a diagnosis of "goutiness" is a refuge for the destitute, a soothing and satisfying phrase to disguise from a patient that we do not know what actually is the matter with him. Slight traumatisms to joints or other parts are readily forgotten, yet they may leave effects behind them, and such after-effects in an elderly person are too readily ascribed to gout. If we cannot quite ascertain the cause of a patient's indigestion, how easy it is to call it gouty! And so on in a hundred other instances. Perhaps it is partly from this that gout has been considered so protean a malady. Until we know the true nature of the disease much more thoroughly than we do at present, this question, like many others, must await its final solution.

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[1] *The Lancet*, January 31, 1903.

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Irregular Gout.

YET on the other hand in those who actually suffer at intervals from articular attacks there do occur a number of morbid phenomena which seem to be a part of their disease and which may in some instances be remedied by the usual measures taken to combat true gout. Eczema is one of these, and so is phlebitis, both of which Dr. Toogood in his experience among the poor considers to be gouty in origin. This writer does not apparently believe in a gouty urethritis, finding that the cases in which this lesion was ascribed by his patients to gout could always be traced to a less abstruse cause. Yet we have met with at least one such case in a member of our own profession who seemed to suffer from a non-specific urethritis which he attributed to gouty inheritance. Bronchitis and a form of asthma also seem to own a gouty origin. What are we to suppose the pathology of these irregular manifestations to be? Is there a deposit of urate of soda in each case in the affected tissue, whether it be the skin, vein, or mucous membrane? Or in the presence of a generally-defective metabolism do other local chemical perversions occur? Or, again, does the gouty state favour the entrance of micro-organisms as does the diabetic? It is easier to ask these questions than to answer them.

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Pathological Summary.

WITH regard to the pathology of gout we may then perhaps sum up our knowledge in a series of propositions: (1) Gout is a morbid condition, of which the most striking phenomena are acute attacks of arthritis, which tend to recur and which are accompanied by redness, pain and œdema of the part. (2) The affected joints are found to be the seat of a deposit of urate of sodium, which occurs first in the articular cartilage and afterwards infiltrates all the surrounding structures. (3) Deposits of this material may also be found in other parts, such as the pinna of the ear, bursæ, &c. In these they may occur without exciting distinct inflammation. (4) Excess of uratic material may be demonstrated in the blood of a gouty patient, the amount being greatest before an acute attack and least immediately after one. (5) The influence of heredity in producing gout is undoubted. Certain poisons, such as lead and malt-liquors, may induce an attack.

We may similarly sum up our ignorance. (1) We do not know how the excess of uratic matter is brought about, whether it is due to increased intake with the food, to increased formation in the body, or to deficient elimination by the excretory organs. (2) We do not know whether the uratic excess is the cause of the phenomena of the disease, or is a mere casual accompaniment of the perverted state of nutrition. (3) Similarly we do not know in the case of the arthritic attacks whether the deposit of urate of soda is the cause or the effect of the local inflammation.

In all probability gout consists in a defect of metabolism whereby products derived from cell-nuclei are not properly used up or excreted. These products may arise either from nuclear matter contained in the food, or from the natural wear and tear of tissue-nuclei: they are retained in the system, and a deposit of one of them—sodium biurate—in the joints sets up irritation there, and causes an acute attack of arthritis.

We must not be blind to the fact that in using "that blessed word" metabolism, we mean no more than the chemical side of life itself. All diseases are disorders of metabolism—in other words, perverted vital activity. Hence we practically come back to our original starting-point—that gout is a disease which we do not understand.

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Reviews of Books.

A Manual of Surgery. By WILLIAM ROSE, M.B., B.S. Lond., F.R.C.S., and ALBERT CARLESS, M.S. Lond., F.R.C.S. Fifth edition. London: Baillière, Tindall, and Cox. 1902. Demy 8vo. Price 21s.

THAT this useful book should have attained its fifth edition so rapidly need cause no surprise. It still holds its place as the most complete and exhaustive manual on surgery in print. Either as a text-book for students or a work of ready reference for the busy practitioner it has no equal. That the book should now commence with a chapter on Surgical Bacteriology and the principles of Antiseptic and Aseptic Surgery instead of a chapter on Inflammation and its treatment is only in keeping with the progress of our knowledge. This part of the volume has had the advantage of being overlooked by such an authority as Prof. R. T. Hewlett. In this edition advantage has been taken of the great progress made in skiagraphy, and it is well illustrated by numerous plates showing the displacements in fractures and dislocations, and the changes occurring in the bones and joints in tubercular disease. Plate XXVI. showing double congenital dislocation of the hip, and Plate XXV. showing tubercular disease of the radius are both excellent examples of the illustrations contained in this edition. The book is beginning to be a little unwieldy on account of its size. The authors should look to it that it loses none of its usefulness from this reason, and should keep it within the bounds of its title, viz., *A Manual of Surgery.*

A Text-Book of Anatomy. Edited by D. J. CUNNINGHAM, F.R.S., M.D. (Edin. et Dubl.), D.Sc., LL.D. (Glasg. et St. And.), D.C.L. (Oxon.). Illustrated with 824 wood engravings from original drawings, many printed in colours. Pp. xxxii and 1309. Young J. Pentland. 1902. Price 31s. 6d.

THIS is a text-book of anatomy by many authors, divided into sections under the following heads :—"The Digestive System," by Prof. Ambrose Birmingham; "The Brain and Spinal Cord, the Respiratory System, and the Ductless Glands," by the Editor; "The Urogenital System," by Prof. Dixon; "Arthrology," by Dr. Hepburn; "The Organs of Sense, and the Integument," by Prof. Howden; "Myology, the Spinal and Cranial Nerves, and the Sympathetic System," by Prof. Paterson; "General Embryology, and the Vascular System," by Profs. Robinson and Young; "Osteology," by Prof. Arthur Thomson; "Surface and Surgical Anatomy," by H. J. Stiles, M.B. It escapes the fault, so often found in such works, of unevenness—the different sections, though not losing the distinctive individuality of each of the authors, are well balanced, giving the impression of being by a single writer.

The book opens with a section on general Embryology, but in addition has a description of the development of the various viscera following their own sections throughout the book. This is obviously of great advantage to the junior student, who thus is not overburdened at the outset with innumerable facts and theories concerning the development of organs, but can assimilate them as he advances, after mastering the main facts of the subject of Embryology. In the same way the descriptions of the various bones are followed by a discussion of their ossification, amply illustrated by coloured figures, which will prove most useful as an aid to memory. The section on Osteology includes also a discussion on Craniology and Morphology. Very useful tables on the nervous supply and movements of muscles are included in the section on Myology. It is a matter for regret, however, that the origin and insertion of muscles are not given with more accuracy of detail. In some cases the description is of the briefest and only in general terms. Undoubtedly the section on the central nervous system is one of the clearest and most concise expositions on the subject yet written. It is admirably arranged in such a way that much which, while of great use to the advanced student, may yet be beyond the beginner, can be omitted by the latter without interrupting the sequence of the description. The same criticism applies to the section of the Cranial Nerves as was made on that of Myology, viz., lack of detail. This is particularly the case in the description of the course of the facial nerve, the intrapetrous course of which is of such great importance, and yet is here dismissed in three lines. The illustrations of the section on the Vascular System are disappointing. more especially those of the arteries, which are flat and lack distance, and are not in keeping with the general tone of the book. It is also to be regretted that the variations of each artery and vein are not arranged so as to come immediately after their respective descriptions instead of being lumped together at the end of the section. Such an arrangement very materially diminishes the value of the section. In the section on the Digestive System an error has been overlooked. In giving the position of the structures traversing the parotid gland, the facial nerve is stated to be deep to the temporo-maxillary vein.

Taken altogether the volume is a most valuable addition to our text-books on Anatomy. Written as it is by some of the leading anatomists of this country who are also teachers, it will have a wide sphere of usefulness not only as a text-book, but also as a work of reference on the subject.

A System of Clinical Medicine, dealing with Diagnosis Prognosis, and Treatment of Disease, for Students and Practitioners. By THOMAS D. SAVILL, M.D. (Lond.). Physician to the West End Hospital for Diseases of the Nervous System, and to St. John's Hospital for Diseases of the Skin, &c. Vol. 1: Local Diseases and Microbic Disorders. Pp. xxiii and 702. London: J. and A. Churchill. 1903. Price 12s. 6d. nett.

ON the appearance of a new textbook of medicine it is natural to ask first, what is the justification for adding to the already existing plethora of such works? The author of the present volume would apparently answer this question by saying that this book is an attempt to inaugurate a new method of presenting the facts of clinical medicine to students of the subject. This "System" does not pretend to compete with such text-books as are associated with the names of Osler or Taylor. It is rather to be compared with such a book as Fenwick's *Medical Diagnosis*, being intended not so much to give a systematic account of the state of our knowledge of medicine, as to teach the student methods of observation and the practical ways of distinguishing between the diseases which he meets with in the course of his hospital work. From this point of view one obvious criticism at once occurs, viz., that a work in two volumes is somewhat more elaborate than the average student is likely to be able to purchase for himself, in addition to his systematic textbooks of medicine, pathology, &c. Further, it seems to us that the author has made the mistake of trying to crowd too much information into the book. He has attempted to make it at once a book of clinical diagnosis and a treatise on medicine in general. He thus falls between two stools. The arrangement adopted is not suitable to supply the whole medical pabulum necessary for salvation, and yet there is so much material crowded in that the tabular and symptomatic arrangement is considerably obscured. The attempt to obviate this by the use of different types makes the book irritating to read steadily through.

The plan adopted is to enumerate first the symptoms characteristic of disease of a certain organ or tract; next to detail the methods of examination available for their study; and lastly to discuss the different diseases *seriatim* in the order suggested by the symptoms or signs. Thus diseases of the heart are classified according as they involve an increased area of præcordial dulness or not, and affections of the lungs as they are, or are not, productive of dulness on percussion. We do not think that in either case the plan works out well. The book gives the impression of being to a great extent a compilation rather than the fruit of the author's own experience. Thus he alludes to the examination of the chest by the Röntgen rays in cases of aneurysm as a mere matter of hearsay, whereas it is an everyday occurrence at any general hospital; and other examples could easily be quoted. Lack of teaching-experience at a medical school is also, no doubt, accountable for a certain want of perspective in the choice of information conveyed. On the whole we think that the volume may be of assistance to students for use just before an examination or as a guide to their wardwork. Much of the information contained in it is valuable, and may serve to recall and arrange the clinical knowledge gained by routine clinical study. Such knowledge, however, if obtained solely from a book, is of no practical use; and this "system" will act as a snare instead of an aid if it lead to any attempt to absorb another's experience ready-made, instead of accumulating a store of first-hand practical knowledge.

Dose-book and Manual of Prescription-Writing. By E. O. THORNTON, M.D., Ph.G. Second Edition, Revised and Enlarged. Illustrated with 2 plates and 27 figures. Pp. 362 London and Philadelphia: W. B. Saunders and Co. 1901.

THIS Manual was prepared for the medical student and gives the composition and strength of all the preparations official in the United States Pharmacopœia. As there is no account of the preparations of the British Pharmacopœia it is of only limited interest to the English student. He may, and probably will, find those portions dealing with the Latinity of prescriptions, the solubilities of chemical substances, and the metric system useful in the course of his studies. Most of us know what the term "shot-gun" prescription implies; but we wonder how many are acquainted with a "Kangaroo" prescription.

The Medical Annual, 1903. A year book of Treatment and Practitioner's Index. By many Contributors. Bristol: John Wright and Co. Pp. 959. Illustrated. Price 7s. 6d. nett.

WITH the present issue the *Medical Annual* attains its majority – in other words, it reaches its twenty-first year—and if it maintains its present high standard of usefulness, it seems likely to reach a hale old age. The volume is divided into three parts, the first being a review by Professor Hare of Philadelphia of therapeutic progress throughout the past year, and including a dictionary of new remedies, and an interesting article on treatment by X-rays, and by high-frequency and high-potential currents: the second and greater part of the work consists of an alphabetically-arranged series of articles on treatment. Most of these are written by well-known authorities and contain a very large amount of useful information. The value of the articles is increased by references to the sources from which they were compiled, and we would suggest that these references might be increased still further with advantage; for although the statement that Dr. So-and-so finds such and such a method of treatment effective is often interesting, it would be of more value if we could verify or amplify the account by reference to the original paper; the name of the authority alone is hardly sufficient for this purpose. Every branch of medical science is included, and many of the articles are made more interesting by excellent illustrations, some coloured, and some reproduced from photographs. The reproductions of skiagrams are particularly good: a languishing belief in the value of X-rays in the diagnosis of renal calculus may well be stimulated by the pictures here shown of phosphouratic and oxalate stones in the pelvis of the kidney. The third part deals with advances in public health and recent legal decisions affecting medical men; both these sections being well written. On the whole we consider that such a book is likely to be very useful to the practitioner; especially to those who cannot find time to separate for themselves the chaff from the wheat in the ever-increasing pile of journals which are bewildering enough even to the man of leisure.

The Work of the Digestive Glands. Lectures by Professor S. P. PAWLOW. Translated by W. K. THOMPSON, M.D., F.R.C.S. Pp. 196 with 19 Figs. Price 6s. nett. Charles Griffin and Co. 1902.

THIS work consists of nine lectures, the first eight of which give a *résumé* of the experimental work carried out in Professor Pawlow's laboratory up to the year 1897; while the last, delivered five years later, contains the results obtained in the interval, and deals especially with the saliva, the bile, and the succus entericus. Professor Pawlow is a modern Ludwig, and with his pupils has carried out the most ingenious experiments on animals which have revolutionised the physiology of the digestive system. The two fundamental features of the digestive secretions are shown to be adaptation to requirements, and secretion in response to a specific stimulus. Thus, sand or other inert substances when introduced into the mouth excite a flow of watery saliva which is all that is required to effect their removal, while eatable substances lead to the secretion of slimy mucin-containing saliva. To show the great advances in physiological knowledge made by Pawlow and his pupils, the subject of the succus entericus may be referred to. It is shown to contain a ferment—enterokinase — which does not act directly on food, but augments the activity of the pancreatic juice, especially its proteolytic function. It may be spoken of as a "ferment of ferments." The specific stimulus which leads to the secretion of enterokinase is the presence of the ferment-constituents of the pancreatic juice in the intestine. The text is well translated by the editor, who has appropriately added a useful note describing Starling and Bayliss' recent work on "Secretin" which is manufactured on the mucous membrane of the duodenum and stimulates pancreatic secretion.

CORRECTION.

THE Secretary of the Association of Medical Men Receiving Resident Patients calls our attention to an error in our notice of their handbook, "*Where shall I send my Patient?*," in which it is stated that the names of the members are given in the book. This is not the case, only the locality of each home and in some cases a pseudonym being given. We are glad to insert this correction, and take the opportunity to express our regret to any members of the Association who may have felt aggrieved at our original notice.

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THE PRACTITIONER.

AUGUST, 1903.

THE TREATMENT OF GOUT IN CARLSBAD.

By B. LONDON, M.D., *Carlsbad.*

BEFORE proceeding to a description of the results of the usual treatment of arthritis, which for decades has been in vogue here, it might, perhaps, be useful to give a short account of the various hypotheses, both ancient and modern, as to the nature of gout; of the results of the most recent investigations concerning its ætiology; and of the most prominent symptoms and course of a disease which is so often to be observed here; and to present an outline of the therapeutic measures which have been recommended in recent years.

As is well known, the advances made in chemistry at the beginning of last century have made it possible for the exact investigations of Andral, Bergmann, Cruveilhier, Murray Forbes, Holland, Parkinson, Petit, Rayer, Pearson, Scudamore, Tennant, Wollaston, and others to demonstrate the fact that, in all probability, gout is induced by an excess of uric acid circulating in the blood and being deposited in the tissues.

Later on, about the middle of the century, Garrod, and still later Bartels, Cantani, Lecorché, and Ranke were able to confirm this theory that the blood of the gouty patient contains an excess of uric acid. Garrod showed, by his famous "thread" experiment, that uric acid was contained in exudation- and transudation-fluids (pericardial, peritoneal, and vesicular) of persons suffering from gout. He was further able to show that, in an arthritic patient, coincidentally with an increase of uric acid in the blood, there is a diminution of this acid in the urine, owing to renal incompetence; and that this occurred several days before an attack of gout. This diminution is said to be more marked during the first few days of the attack, after which the amount gradually approximates to the normal.

In chronic gout, on the other hand, the uric acid in the urine of an arthritic patient remains permanently diminished.

Bouchard was of opinion that the association of typical gout with many other diseases and functional disturbances of various organs, more particularly with other metabolic disorders, must be looked upon as the expression of a faulty constitutional disposition, that is, of an arthritic diathesis (*arthritisme*). Bouchard endeavoured to ascribe the basis of gout to a retardation of the oxidative processes in the organism, and indeed he looked upon uric acid with diminished alkalinity of the blood as a product of incomplete oxidation. He thought that abnormal acidity of the blood diminished the solubility of uric acid, and favoured the deposition of acid urates.

In the various diseases and functional disorders, which occur in association with gouty changes, or in persons affected with hereditary arthritis, Lecorché recognised the toxic effect of uric acid in the circulation, such uric acid being produced in excess, imperfectly oxidised, or insufficiently excreted, thus increasing the domain of the uric-acid diathesis.

Haig believes the influence of uric acid to be much greater, inasmuch as he considers the most formidable diseases, such as migraine, dyspepsia, hysteria, asthma, Raynaud's disease, even epilepsy, paroxysmal hæmoglobinuria, psychoses, heart-affections, &c., &c., as sequels of " Uricacidæmia."

Cantani looks upon the uric-acid dyscrasia in gout as the result of a localised disturbance of nutrition in the articular cartilages and periarticular tissues. Charcot, Murchison, Fothergill, Latham, Harley, and others considered gout as the result of a functional disturbance of the liver. Ebstein maintains that the principal seat of the formation of uric acid in gout is in the muscles and bones, as regards the localised processes of uric-acid deposit. Senator attributes the chief rôle in the formation of excess of uric acid to the spleen, the functions of which are disturbed by abnormal nourishment. A few years ago Dyce Duckworth was strongly of opinion that arthritis represented a primary neurosis.

With regard to the general storing up of uric acid, Levison holds the view that a constant retention of even minute quantities of uric acid suffices to explain the manifestations of gout, and postulates a granular degeneration of the kidneys as an antecedent condition. He points out that an early-occurring functional disturbance of the kidneys in this disease must not be considered as a chronic interstitial nephritis, because, pathologically and histologically, the degeneration commences in the convoluted tubules, and is followed by a glomerulitis and overgrowth in the interstitial connective tissue.

Ebstein has partly modified Garrod's theory of the origin of gout. He maintains that the local mischief which produces the gouty necrotic areas is not due to a deposition of crystalline acids, but to the presence of neutral urates dissolved in the tissue-juices. He has investigated anatomically the necrotic foci which surround all uratic deposits, and he comes to the conclusion that the circulating uric acid occurs in the form of soluble neutral urates, constituting an aseptic poison, which causes necrosis of certain tissues, into which the acid urates then crystallise out. The crystallising-out of these acid urates is said to be produced by the formation of free acids in the necrotic areas. He ascribes the formation of these typical necrotic areas in gout to a storing up of uric acid; and holds that chronic and visceral gout arise from a general storing up of uric acid in consequence of renal insufficiency. Besides this, a disturbance of the movement of fluids in the neighbourhood of the points of origin of uric acid leads to a localised storing up of uric acid, which is responsible for the acute joint-affection.

Roberts bases his theory upon the following hypothesis:—He believes that uric acid exists in the blood and in the urine in the form of quadriurates. These are capable of conversion into biurates in the presence of alkaline carbonates and phosphates. Whilst, normally, quadriurates can be excreted unchanged by the kidneys, in gouty conditions, as a consequence of disturbed influences, these salts remain unusually long in the blood, and become there converted in the presence of large quantities of sodium carbonate into bi-urates. These are insoluble in the blood-serum, and are therefore with difficulty excreted in the urine; hence their crystalline deposition in an attack of gout.

I must finally mention the interesting theory of Pfeiffer. In a healthy person the uric acid which is produced in the blood and in all the tissues is capable of being disintegrated by the viscera, with the exception of the kidneys. This depends upon the capacity of all organs to decompose, as well as to build up, uric acid; so that the organs and the blood of a healthy man do not contain uric acid, whereas its presence in the blood and organs of a gouty person is constant, and depends upon a diminution or abolition of this capacity for decomposing uric acid. This undecomposed uric acid appears in the various organs, but more particularly in the urine, in an insoluble or, at any rate, difficultly-soluble modification. It produces in the tissues the well-known uratic deposits and tophi, and in the urine the sediments of crystalline uric acid (*Sedimentum lateritium*). In the early stages of gout, the quantity of uric acid excreted in the urine is diminished, as compared with the normal, and only in some chronic cases with considerable uratic deposits do we find an increase of uric-acid production and uric-acid excretion. The quantity of uric acid, therefore, as compared with the normal is unchanged, only the quality being altered—that is to say, it enters into insoluble combinations or becomes imperfectly soluble.

It is not the insoluble or difficultly-soluble modifications of uric acid which cause pain and irritation, but only those soluble modifications which appear in any tissue in abnormal quantities or enter into pain-producing combinations. As long as the usual form of uric acid, that is to say, the double salt of sodium urate and sodium phosphate, is contained in a normal amount in the tissues, no irritating effect is produced upon them; whereas painful sensations and irritation are produced by an amount of this combination which much exceeds the normal. Sodium biurate uncombined with sodium phosphate is responsible for the production of the greatest pain. During an attack of acute gout it is usual for a considerable increase in the excretion of uric acid to take place, and at the same time the uric acid appears in the urine fully combined. It is therefore highly probable that an acute attack arises in consequence either of the production of large quantities of soluble uric acid, or from the fact that stored up masses of uric acid in insoluble or slightly-painful form are brought into solution, and so give rise to pain and irritation.

The chronic changes in gout, such as uratic deposits and tophi, arise from the urates of sodium contained in the tissues being rendered insoluble by carbonates. As carbonates exist everywhere in the body, nothing further is required for the production of the needles of sodium-biurate than that the amount of soluble uric-acid compounds present in the fluids should exceed the percentage at which the biurate of sodium is capable of remaining in solution in the presence of salts of carbonic acid. If these conditions for the separating out of sodium urate are not present, numerous attacks of acute gout may occur without leaving traces of disturbance; on the other hand, we see in chronic gouty attacks changes of a chronic nature occurring, which are not referable to the deposition of biurate of sodium, but are dependent upon the irritating effect of excessive amounts of uric acid, which are permanently present in the tissues.

Thanks to the ceaseless energy exhibited by Kossel, Miescher and Emil Fischer, in their clinical and experimental investigations during the last few decades of the nineteenth century in the domain of gout, we may reasonably expect a more intimate acquaintance with the rôle played by uric acid in the human organism and with the pathogenesis of gout. One advance in this subject is the recognition of the origin of uric acid in the organism of mammals, and its relationship to the substance of the cell-nucleus. This has been brought about by the investigations of Kossel into the decomposition-products of nucleic acid, which was discovered by Miescher.

A further advance consists in the invaluable discovery of the chemical structure of uric acid and of its allied bodies, which has resulted in the brilliant investigations of Emil Fischer on the subject of purin and its derivatives, viz., hypoxanthin (oxypurin), xanthin (dioxypurin), and uric acid (trioxypurin).

It is true that these investigations have up to the present produced no unassailable result. But we can already assume with the greatest confidence that the presence of purin-bodies is not so much responsible for the production, oxidation, excretion, or precipitation of uric acid in the organism as the behaviour of the remaining atom-complexes with which the purin-substances circulating in the organism happen to be combined.

In spite of all careful and exact investigations, it has not yet been certainly determined in what combinations uric acid circulates in the blood and in the tissue-juices. Minkowski

(Leyden's *Handbuch der Ernährungstherapie*, Band II. S. 507) believes that the combination is a more or less loose one with a complicated atom-complex, in which the uric acid circulates in the blood and by means of which its ultimate destiny in the organism is determined. At first he considered that nucleic acid might exist in the organism as a carrier of the purin-bases in combination with uric acid. He was able indeed to show (*Verhandlungen des XVIII. Congresses für innere Medizin*, 1900), that under certain experimental conditions uric acid was able to enter into combination with nucleic acid in a form in which it could not be precipitated by the usual reagents and even remained soluble in an acid medium. If an alkaline solution of pure nucleic acid is brought into contact with a solution of sodium urate the precipitation may be prevented by acetic acid, or by a mixture of an alkaline solution of silver and magnesia.

It appears also that nucleic acid may take up uric acid in its molecule, and trioxypurin resembles in this respect the other purin-bases, which Kossel and Schmiedeberg have shown to be capable of attaching themselves in considerable quantities to nucleic acid. From these premises Minkowski infers that uric acid can occur in the blood and in the tissue-juices like the other purin-compounds, first of all in combination with nucleic acid; and that in consequence of this association the conversion of purin-bases into uric acid may take place, and that the solubility and distribution, together with the final destiny, of uric acid in the organism may be determined.

The uric acid in the urine occurs in much greater concentration than in the blood. For the rest we are in nowise clear as to the conditions which produce the solubility or the precipitation of the uric acid which has been excreted. With regard to the uric acid in the urine of an arthritic patient. Various investigators, as already mentioned, such as Bartels, Bouchard, Cantani, Ebstein, Goto, His, Magnus Levy, Lecorché, Pfeiffer, Sprague, have, as a result of countless experiments, been able to confirm more or less the statements of Garrod, viz.:—

1. That daily excretion of uric acid in an arthritic patient during an interval of immunity is approximately equivalent to that in a healthy person.

2. Further, in chronic gout, even in those cases associated

with copious uratic deposits a constant variation of the normal amount of uric-acid excretion in any particular direction cannot with certainty be demonstrated.

3. On the other hand, it appears to be certain that the occurrence of an acute attack of gout is associated with definite variations in the excretion of uric acid, and, indeed, a diminution in the amount of uric acid in the urine appears immediately to precede an attack, only to be followed, in the course of the attack and after it, by an increase in the uric-acid excretion. These variations in the excretion of uric acid must evidently be of considerable importance in the pathogenesis of gout.

The diminution in the excretion of uric acid which precedes an attack may depend upon diminished uric-acid production or upon disturbed processes of renal secretion. Further, it is possible that the local retention of urates or of their immediate predecessors in the affected joints may be the cause of the diminished excretion of uric acid in the urine. The increased excretion of uric acid after an attack may depend, as well upon increased uric-acid production as upon subsequent elimination of uric-acid masses which had been previously retained in the organism.

Finally, the variations in the excretion of uric acid may be dependent upon the fact that a variable portion of the uric acid which has been produced undergoes further decomposition in the organism. The significance of the excretion of uric acid in the urine of a gouty patient can only be apparent when at the same time the amount of uric acid in the blood is taken into consideration. In his theory of gout, Garrod showed, in addition to the fact that the gouty deposits consist of sodium mono-urate, that the blood of an arthritic patient always contains uric acid in abnormal quantity. Furthermore, it is by no means clear what significance is to be attached to the increased amount of uric acid present in the blood in gout.

Klemperer believes that the overloading of the blood with uric acid is certainly not the only cause of the precipitation of urates in the gouty areas. It is much more likely to be the local changes occurring in those areas of uratic deposits which are responsible for the production of such deposits.

We know, in addition, that in other diseases, such as nephritis, lead-poisoning, arteriosclerosis, pneumonia, and leuchæmia, a large amount of uric acid is present in the blood. It has, however, been shown that arteriosclerosis, lead-intoxication, and nephritis possess associations with gout.

Again, Weintraud has been able to demonstrate a considerable, though transitory, amount of uric acid in the blood, even in healthy people, as a result of the ingestion of sheep's thymus, which is very rich in nuclein-substances. As regards the cause of the production of an increased amount of uric acid in the blood of an arthritic patient, most experiments on this subject have, up to the present, shown that this increase results from a defective excretion of this substance by the kidneys.

With regard to the special rôle played by nucleic acid and its derivatives in the solution, distribution, and final destiny of uric acid, it is probable that nucleic acid must be to some extent responsible even for the anomalous changes in uric acid that take place in arthritis. According to this, the fundamental disturbances in gout are to be sought for, as Minkowski maintains (*Die Gicht*, 1903), in certain abnormal metabolic changes in the cell-nuclei, bearing in mind that probably these abnormal processes are dependent, not so much upon the members of the purin-group itself, as upon the complicated atom-complexes which are combined with it.

ÆTIOLOGY.

Formerly it was almost looked upon as an axiom that gout was caused by an insufficient oxidation of the uric acid which originated in albuminous substances, inasmuch as this insufficient oxidation of uric acid brought about a diminution in the alkalinity of the blood owing to an accumulation of acid products of metabolism. At the present time, however, the results of the most recent investigation teach us that we must look for the ætiological factor in gout, either in the influence of nuclein-decomposition in the intestinal walls, in association with the processes of digestive leucocytosis, or else in the special changes produced in the intestinal contents. Here the fate of the purin-bases contained in the intestine may be influenced by abnormal disintegration-processes, or noxious bodies may be produced which are liable to favour the production of gout.

According to all experience a predisposition to gout in an individual is born in him; and it is doubtless hereditary. Besides this inborn diathesis, I can positively affirm, from the experience obtained from my numerous arthritic patients in Carlsbad, that a most important predisposing factor is luxurious living, particularly in the excessive use of meat and alcohol (especially champagne, port, sherry, and beer), and insufficient exercise. I have further had an opportunity of observing among the poor, many of whom are unable to obtain any luxuries, instances of arthritis, not only of the irregular character, but also of the typical form of gout.

According to age, the youngest individual among my patients was 14 years old (a boy), and the oldest 65 years of age (a man).

Among the adventitious causes mentioned by the patients as determining an attack of acute gout, were errors of diet, exposure to cold or wet, powerful emotions, sometimes local traumatism or general shock to the system, damp, and cold winds; bad weather in the spring and autumn appeared especially to have an injurious effect upon gouty patients.

Finally, there appears to be a peculiar relationship between chronic lead-poisoning and gout, inasmuch as in an existing gouty condition the effect of chronic lead-intoxication, as in painters, type-founders, &c., not infrequently determines an attack of gouty symptoms.

Garrod, Charcot, Potain, Lancereaux, Sir Dyce Duckworth, Senator, Magnus Levy, Lüthje, and others, have appreciated the significance of chronic lead-poisoning in the development of gout. In the cases that I have observed, I was able to remark not only the special arthralgias which occur in lead-cachexia, but also the specific and characteristic symptoms of gout. Most of these people were affected with multi-articular gout. In lead-gout are to be found the characteristic anatomical changes in the kidneys, which are produced by the absorption of lead, resulting in the so-called "saturnine contracted kidney." Garrod and Bouchard found in cases of gout due to lead an abnormally high amount of uric acid in the blood with a diminished excretion of the same in the urine.

During an experience of nearly 30 years in Carlsbad I have had considerable opportunity of collecting plenty of material from gouty cases. I was careful to study these with regard to their geographical distribution, and I found in this respect great differences between the various countries. The greatest number of arthritic patients came from England, Holland, and the United States of America. Fewer patients came from Russia, Sweden and Norway, Denmark, Germany, and France. The south of France, Italy, Spain, Turkey, and Greece supplied the smallest number. Those patients who came from India and Egypt were nearly always Europeans who had lived there as *bons vivants*, leading a sedentary life, many of them possessing a hereditary history of gout. They were mostly robust, well-nourished people, suffering from typical regular gout; irregular gout being more particularly observed among weakly and cachectic individuals.

In the course of a 30-years' practice which I have conducted in Carlsbad, I have frequently had occasion to observe that in those individuals who had formerly suffered from general gout, or from a local attack of gout, an infective septic suppurative arthritis has resulted, not infrequently either immediately after an attack of pneumonia, or coincidentally with it. The same has occurred after a number of other diseases, such as pleurisy, peritonitis, pericarditis, meningitis, typhoid fever, influenza, scarlatina, dysentery, erysipelas, as a result of the invasion by the bacilli or cocci (associated with these diseases) of joints, more particularly those affected with gout. Clinical as well as bacteriological investigation has demonstrated that this form of joint-disease occurs much more frequently in arthritic patients than is generally believed. As a result of these experiences I can conscientiously endorse what Howard Marsh maintains in his Bradshaw Lecture on Septic Arthritis, namely, "if a patient previously have suffered from gout so that his joints were already damaged by the gout, they lose their normal resistance, and thus become localities in which the irritative action of the bacilli and cocci, or their toxins, can take effect."

PATHOLOGICAL ANATOMY.

The precipitation of uric-acid salts in the tissues of the joints usually takes place symmetrically on both sides of the body, and often affects a large number of joints without the appearance of external tophi. As already explained, a gouty precipitation, especially in the first attack, localises itself in the metatarso-phalangeal joint of the great toe. This seat of election may possibly depend upon the imperfect circulation in tissues far removed from the heart, or it may depend upon the relatively heavy work thrown upon this joint, in consequence of its bearing the whole weight of the body.

Not uncommonly many joints are involved in an acute attack of gout. It often happens that one articulation after another is affected, so that hardly any joint is safe from attack. Even the sterno-clavicular joint is not spared. Tendons become involved, and isolated gouty inflammations occur in the tendon-sheaths, particularly in the tendo Achillis, as well as in the extensor tendons of the hand and foot. The gouty deposits affect primarily the articular cartilages, then the synovial membranes and articular fibrous capsules; later, the tendon-sheaths and bursæ are affected, and even the periosteum of the epiphyses is involved. In a recent case the inner aspect of the joint-capsule looks as if it were dusted with sugar. Under a high power these deposits have the appearance of needle-shaped crystals, arranged sometimes side by side, at other times in the form of sheaves, or again star-shaped; chemically, these crystals are composed of biurate of sodium, together with sodium chloride, calcium carbonate, calcium phosphate, and sometimes also hippuric acid.

In recent times, Ebstein has carried out the most accurate histological investigations on the deposits in articular cartilages. With the aid of delicate staining-methods, and by the use of polarised light, he investigated the hyaline articular cartilages of gouty patients, and was able to demonstrate that the uratic deposits are surrounded by portions of tissue which have unquestionably undergone necrotic degeneration. And he convinced himself that these necrotic areas were primarily produced by the uric acid in the tissues, and that the uratic precipitation subsequently took place in the destroyed areas. Inasmuch as these necrotic foci with their crystalline deposit react as irritants upon the surrounding tissues, various forms of inflammation are brought about. Thus, thickening of the synovial membrane, of the capsule and of the periarticular connective tissue gradually takes place; the cartilages become loose and disintegrated; the epiphyses become carious; joint-deformities, dislocations, and even ankyloses supervene.

The variety of the anatomical lesions which can be brought about in the joints by gout explains why so many peculiarities are observed when gouty joints are X-rayed. X-rays pass more easily through uric-acid salts than through the calcium-salts of normal bones. The uratic deposits in joints appear, therefore, as lighter spots, while on the other hand the exostoses throw darker shadows.

Of all the visceral manifestations of gout, those in the kidneys present the most frequent anatomical changes, inasmuch as they exhibit the specific stamp of their gouty nature in the precipitation of uric-acid salts. A granular degenerative affection of the kidneys is most often observed in gout. Such a kidney is known as gouty or contracted. The uratic deposits are chiefly found in the papillary zone, whereas the induration usually affects the cortex of the kidneys. Sometimes the shrunken kidney of the arthritic patient completely resembles the granular kidney of alcoholism or saturnine poisoning. There is often an associated arterio-sclerosis. Some cases exhibit no other gouty manifestations, only a gouty kidney, with uratic deposits and circumscribed necrotic areas being found.

According to Ebstein's view, this is a primary localisation of gout in the kidneys (primary kidney-gout) which occurs long before the joints become involved. The further progress of kidney-gout results in albuminuria with the appearance of hyaline and granular casts, tubular epithelium and leucocytes.

Besides granular degeneration of the kidneys, there is a close association between nephrolithiasis and gout. The gouty deposits bring about uric-acid concretions. Emphysema and hepatic cirrhosis are frequently found *post mortem.*

SYMPTOMATOLOGY.

The first and most characteristic symptom of a gouty attack is a sudden and painful affection of a joint. Very often such an attack supervenes suddenly and without prodromata, but more frequently there are characteristic warnings, such as depression of spirits, aches and pains, uncomfortable sensations in all organs and parts of the body, digestive disturbances, and increased or diminished excretion of sweat. Immediately before an attack existing nervous affections, such as migraine, asthma, or neuralgia, suddenly disappear. Such phenomena may recur periodically, and may represent the main symptom or prototype of regular or typical gout, in contradistinction to those forms in which pronounced symptoms of acute gout are absent, or at any rate not clearly recognisable, and which manifest themselves by severe disturbances of other organs, particularly internal ones, and are designated as irregular, atonic, latent, visceral, or metastatic gout.

After the first attack months, or even years, may elapse before another supervenes, and this takes place most frequently in the spring or autumn. As already mentioned these attacks affect the metatarso-phalangeal joint of the great toe, but later nearly all other joints become involved.

Swellings often appear in the neighbourhood of joints as well as in the cartilages of the ear. These are aseptic, inflammatory, circumscribed, and often fluctuating, and commonly pass into tough gouty nodules or tophi. They often originate in the bursæ in the neighbourhood of joints.

Such tophi seldom diminish in size by absorption; they more frequently increase as a result of recurring gouty attacks, and often produce ugly deformities of the joints. Often, however, an old tophus becomes absorbed in consequence of recent inflammatory exudation succeeding a renewed gouty attack, exerting a softening effect upon the deposit.

Sometimes a softened nodule covered by a thin layer of skin perforates as a result of trauma or ulceration. A thin milky fluid then escapes, in which numerous uratic crystals are suspended; or a tough, thick, viscid, silky mass may be extruded, which consists microscopically of fine needle-shaped crystals. The presence of uric acid in such tophi can be demonstrated by the murexide test. The addition of an acid shows under the microscope the conversion of the fine needle-shaped crystals of sodium urate into the rhombic crystals of free uric acid.

The aseptic nature of an arthritic inflammation is demonstrated by the fact that it rarely suppurates. Suppuration of gouty joints always occurs as the result of a secondary infection.

A characteristic feature occurring with the disappearance of the attack and, as a rule, after the inflammatory swelling has gone down, is a desquamation of the epidermis, often even a shedding of the nails.

The subjective symptoms manifest themselves in an acute attack of gout, by disturbed sleep, the result of pain; and psychical depression and nervous irritability. Objectively, we observe an increased temperature, of a remittent or intermittent character; frequently, however, particularly in multi-articular gout, the temperature remains raised, and exhibits irregular oscillations.

Palpitation of the heart with irregularity of the pulse is often present, to disappear as the attack is passing off. Where arteriosclerosis and myocarditis are present an acute attack of gout is sometimes the immediate forerunner of serious cardiac disturbances. I have often observed tracheal and bronchial catarrh associated with an attack in gouty patients.

Arthritic patients in Carlsbad have most frequently complained of indigestion. There are many factors standing in a causal relationship to gouty dyspepsia. Among these we must, of course, include such drugs as colchicum, sodium salicylate, liqueur de Laville, &c., which are used in the treatment of gout, and are liable to produce disturbances of digestion. As already mentioned, dyspepsia may be a direct result of an arthritic dyscrasia, representing a special form of visceral gout. Numerous observations have most certainly shown that affections of the organs of digestion may be produced by uric acid, or by closely-related chemical compounds. According to Hayem and His the most various digestive disorders may be produced by a transient precipitation of urates in the walls of the alimentary canal.

I have observed cases of gouty dyspepsia in Carlsbad with remarkable frequency. My investigations into the contents of the stomach revealed no constant results. Sometimes I found a diminution of hydrochloric acid; at other times the hydrochloric acid was completely absent, its place being taken by lactic acid. Sometimes there was diminished acidity, sometimes hyperacidity. Atony of the stomach or intestines was often present. Frequently, diminished motility with active fermentation was to be observed. Also it was not uncommon to find the stomach-symptoms alternating with an attack of gout.

Bouchard considered the slighter forms of gouty dyspepsia as masked arthritis, and characterised the severe affections of the stomach as metastatic gout. In the majority of the gouty patients whom I have had an opportunity of treating in Carlsbad there was present a chronic and obstinate constipation which frequently alternated with stercoraceous diarrhœa, or with nervous diarrhœa. Not infrequently symptoms of membranous colitis occurred. I have often observed instances of intestinal auto-intoxication with a remarkable rise in the excretion of indican. These patients were for the most part the subjects of mental depression and hypochondriasis. I found that the majority of gouty patients suffered from hæmorrhoids.

I have not infrequently observed among gouty patients in Carlsbad cases of psoriasis linguæ. Often, too, in arthritis, as well as in diabetes, alveolar periostitis has been present, resulting in the falling out of otherwise sound teeth.

A chronic pharyngitis is often to be observed which is usually very intractable to treatment, and uratic deposits occasionally occur in the mucous membrane between the tonsils and the uvula. The same noxious elements which cause inflammatory conditions of the joints and other organs may be responsible for functional disturbances, or even inflammatory changes in the mucous membranes of the stomach and intestines.

With few exceptions, my gouty patients, during the acute attack, presented a transitory hepatic enlargement. Palpation of the liver was painful, and disturbance in the secretion of the bile often present. Associated with affections of the organs of digestion, functional disturbances of the circulation and neuroses of the heart and blood-vessels were often observed.

Of all gouty diseases of the viscera affections of the urinary organs stand preëminent, in that they show a close relationship to arthritis; and, as stated above in the description of the anatomical changes in the kidneys, they bear the specific mark of their gouty nature in the deposits of uric-acid salts.

In the later stages of gout albuminuria usually supervenes with hyaline and granular casts, renal epithelium and leucocytes. The uratic deposits in the kidneys not infrequently induced a nephrolithiasis, leading to the discharge of sand and gravel, and, as a complication of this, resulting in pyelonephritis, hydro- and pyonephrosis.

Many authors look upon the hæmaturia which occurs in consequence of renal calculi as equivalent to an attack of gout. The hæmaturia supervenes suddenly and renders the urine of a dark-red colour; occasionally it resembles pure blood. In most of these renal hæmorrhages (known as gouty hæmaturia, or congestive hyperæmia of the kidneys), there is a total absence of the signs of nephrolithiasis or of contracted granular kidney. The onset of the hæmorrhages is sudden, without any apparent cause, and recurs from time to time in the same manner. Sometimes they are preceded by dragging pains in the loins, which appear to be associated with the discharge of a considerable quantity of blood. Such bleeding from the kidneys may last for days or weeks, without the supervention of albuminuria or the evacuation of concretions, and may disappear without leaving any trace of renal disease.

Simple functional disturbances or catarrhal inflammatory affections of the renal pelvis, of the bladder, and of the urethra, appearing suddenly, may be quite independent of the presence of concretions, and are referable to the influence of gout or of gouty urine upon the mucous membrane of the urinary organs. The same is true of bladder-irritation associated with polyuria, pyelitis, cystitis, and urethritis urica.

An interesting complication has been frequently observed, namely, a urethritis, strikingly like gonorrhœa, without the presence of gonococci; and this occurred in arthritic patients in whom a gonorrhœal infection has never been present. The arthritic nature of the disorder was evidenced by its course, by its appearance immediately before an attack of gout, and by its spontaneous disappearance after the articular trouble was over.

I have not infrequently met with cases of orchitis and epididymitis here. The left testis was most frequently affected. It was not possible, however, to detect uratic deposits in these cases. The orchitis usually preceded the joint-affections and disappeared when these occurred; it frequently accompanied the gouty attacks or was intimately associated with them. Elderly patients often complained of painful micturition or of lancinating pain in the testes and perineum. Rectal examination revealed an enlarged and painful prostate.

In advanced stages of gout, the heart and vascular system exhibited just the same circulatory disturbances as are referable to excesses in eating and drinking and smoking, to syphilis and lead-poisoning, viz.:—atheromatous degeneration of the blood-vessels and endocardium, and arteriosclerotic changes. Cardiac hypertrophy or myocarditis with feeble and arrhythmic action are responsible for the frequent complaints of palpitation, cardiac asthma, and angina pectoris. Mitral stenosis and aortic regurgitation are not seldom present.

Arteriosclerosis seems to occur earlier in arthritic patients, the seat of election in gout being the coronary arteries; a portion of the cases of sudden death occurring among gouty people would seem therefore to be due to such cardiac mischief. No doubt also, the analogous changes in the cerebral vessels leading to hæmorrhages and softened areas must be responsible for other cases of sudden death.

In consequence of a disposition to venous congestion and phlebo-sclerotic changes due to abnormal constitution of the vessel-walls, arthritics are very subject to varices, phlebitis and venous thrombosis of the lower extremities.

The gouty metastases affecting the heart (as they were formerly called) are really fatal attacks of angina pectoris, which, as a special form of abnormal gout, have been described by Payel and Tuckwell; as well as severe cardiac asthma with œdema of the lungs and embolism of the pulmonary artery.

Many and various are the respiratory troubles of gouty patients. Post-nasal and bronchial catarrh are very common and intractable. Emphysema often supervenes.

Hay-fever appears to be related to gout, at any rate, in so far that a neuropathic predisposition is present in both. The same may be said of asthma. A long-continued and obstinate bronchitis often occurs, and not uncommonly accompanies the circulatory and renal affections. Œdema of the lungs is often associated with kidney-disease and sclerosis of the coronary arteries.

Pleurisy and pneumonia appear to be direct sequels of gout. Brissaud believes that regularly-recurring typical attacks of gout are often replaced by pneumonic affections. Potain called attention to the favourable effect of colchicum in gouty pneumonia.

I must finally recall the fact that tubercle and gout are seldom met with in the same person. Phthisis, in an arthritic patient, tends to a slow development of tissue-shrinking and scarring; but the one disease does not exclude the other.

According to the published observations of Charcot, Dyce Duckworth, Lecorché, Ollivier and other careful clinicians, it would appear that the nervous system may be affected by the specific gouty changes. Thus, neurasthenia with its protean manifestations, is often met with, particularly mental depression and hypochondriasis. Many authors assert a relationship between an excess of uric acid in the blood and epilepsy. Bayle, Garrod, Lorry, Lynch and others have observed psychoses, particularly maniacal manifestations appearing after the sudden subsidence of articular gout, which are looked upon as relapses, or the phenomena of masked gout, and which vanish with the onset of a regular attack of gout. It must be mentioned that psychoses, which are ascribed to the influence of gout, are sometimes (especially the maniacal forms of toxæmic psychoses) referable to encephalopathia alcoholica or saturnina and to chronic uræmia.

Arteriosclerosis and contracted kidney pave the way to hæmorrhagic apoplexy. According to Gowers, gouty deposits in the spinal meninges are the cause of acute and sub-acute myelitis.

Those who often have the opportunity of treating cases of arthritis in its most varied forms must have remarked that affections of the peripheral nerves, particularly the various forms of neuralgia, frequently manifest themselves long before the appearance of the first typical attack of gout; or they may alternate with these, or accompany them. Buzzard, Duckworth, Ebstein, and others look upon these peripheral neuralgias occurring in arthritic subjects as a true neuritis.

Among the large number of cases which I have seen in Carlsbad must be mentioned a remarkable association of gout and glycosuria which has so often been observed. Investigation of these cases revealed the interesting fact that most of them possessed a family history of gout, and that they had suffered from arthritis before the onset of the glycosuria. I have observed many cases of diabetes complicated with arthritis, nephrolithiasis, and obesity. Fleiner and Hoppe Seyler attribute the gouty glycosuria to a sclerosis of the pancreatic arteries inasmuch as arteriosclerosis is so common in gout.

Another interesting feature which I ought to mention, is that, of several members of the same family, I have observed some to be affected with gout only, others with glycosuria alone, and others with obesity alone. I cannot look upon this striking fact as merely the result of coincidence, but prefer to regard it as showing a close relationship between these diseases.

Among the various eye-affections which I have noticed among my patients here, I may mention glaucoma, keratitis, hæmorrhagic iritis, and the characteristic forms of conjunctivitis met with in gout.

As before mentioned, the skin and subcutaneous connective tissue are the favourite seats of specific gouty uratic deposits. Among the various forms of skin-disease referable to gouty dyscrasia as a predisposing cause, I have observed *obstinate and oft-recurring cases of eczema in its most varied manifestations.* As ætiological factors may be considered an excess of uric acid in the blood, or those errors of metabolism which are the basis of gout, or an intestinal auto-intoxication. I have seen in the same individual patches of psoriasis with or without eczema distributed over the extensor and flexor surfaces of the limbs, and indeed over the whole body.

Acne, furuncles, and carbuncles were present mostly in glycosuric or nephritic subjects; in a large number of cases, I have noticed a skin-irritation (*pruritus cutaneus*) as well as *pruritus ani et vulvæ* (without glycosuria and hæmorrhoids), together with numberless cases of urticaria. In all these cases the urine exhibited an excess of indican.

In a few cases I have noticed an œdema of the skin in those suffering at the same time from intestinal dyspepsia. The œdema was circumscribed, and appeared on the nose, cheeks, eyelids, and forehead, only to disappear again in a few hours. It corresponds with the "*nodosités cutanées éphemères des arthritiques*" described by Lecorché, Fereol, Graves, and others.

DIAGNOSIS.

Numerous observations and experiences leave no doubt that rheumatism of joints, of long standing, predisposes these articulations to subsequent attacks of gout. If any chronic traces of rheumatic joint-disease persist, these may become incorporated in the gouty changes that ensue. Thus, from a clinical and pathological point of view, well-known mixed forms may exist under the name of "rheumatic gout" or "gouty rheumatism."

Arthritis deformans, although possessing no ætiological relationship with true gout, may be, on a superficial examination, erroneously confounded with arthritis urica. But arthritis deformans occurs usually in badly-fed and weakly persons (particularly among women). The disease is mostly multiarticular, beginning in the small joints of the hand, and extending gradually to the larger joints; being almost always associated with destruction and ossifying hyperplasia of the cartilages.

Finally, I must mention Heberden's nodes, which depend upon fibrous and osseous outgrowths in the phalanges, and about the association of which with arthritis there was much controversy. Pfeiffer, Potain, and Duckworth have recently proved the gouty nature of this affection.

PROGNOSIS.

So far as the prognosis in gout is concerned, I have satisfied myself that this is, in general, the more favourable in proportion as the attacks maintain their regular character. The attacks gradually occur after longer intervals, and finally cease altogether. As soon as persistent changes in, and crippling of, the joints occurred, visceral disease made its appearance, and the prognosis became doubtful. In such cases the ultimate fate of the patient hangs upon the state of his heart and kidneys.

(*To be continued.*)

SOME POINTS ON METABOLISM IN GOUT:

WITH SPECIAL REFERENCE TO THE RELATIONSHIP BETWEEN THE URIC-ACID AND THE PHOSPHORIC-ACID ELIMINATION IN THE INTERVALS AND DURING ACUTE ATTACKS.*

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[Charts 1 to 3.]

FOR a number of years I have been interested in the study of the metabolism in gout, and some points of interest have developed which have led me to publish the following notes. While following the urine of a case of chronic gout with tophi during the early part of 1898, I was struck with the marked parallelism between the uric-acid and phosphoric-acid curves during the quiescent periods and during acute attacks. This case was not reported, but the chart (Case I., Chart I.) was reproduced in the third edition of Dr. Osler's[1] text-book, published in 1898. Subsequent study has led me to the conclusion that this parallelism is a fairly constant one in gout. This intimate association, further, seems to throw some light on the possible source of uric acid.

There has been a tendency in recent years to minimise the importance of uric acid in the ætiology of gout. Whether there is sufficient justification for this scepticism is questionable. One thing is quite certain, and that is that there is a great difference between the amount of uric acid excreted in the quiescent intervals and during the acute attacks of arthritis. In a number of our cases of chronic gout with tophi I have found that, in the intervals, the excretion of uric acid is remarkably small, often falling below 0·2 gram, and even below 0·1 gram in the twenty-four hours. Then in a day or two after the onset of an acute arthritis, the uric acid gradually rises, until it ranges above 0·4 gram, the accepted lower limit for normal. It may reach 0·7 gram, the normal average, or even as high as 1.0 gram, or higher, although

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* From Prof. Osler's Clinic at the Johns Hopkins Hospital, Baltimore.

0·725 gram has been the highest point reached in any of our cases. The uric-acid curve remains within normal limits for a few days, and then, with the subsidence of the acute manifestations, it falls below normal, and there is a diminished elimination until after the onset of another acute attack. This is well brought out in Table I. and Chart I., where the curve during one entire acute attack and parts of two other acute attacks is represented.

This case also shows very strikingly the intimate relationship between the phosphoric-acid and uric-acid curves. The phosphoric-acid excretion was always far below 2·5 grams, the average daily excretion, according to Hammarsten, but was much lower during the quiescent intervals, rising parallel with the uric-acid curve during the acute attack and again falling with the subsidence of the acute symptoms (Case I., Table I., Chart I.). This intimate association between these two acids seems to point towards a common cause and to suggest a common source. There is now abundant evidence, which will not be given in detail here, pointing to the probability that uric acid is largely derived from nuclein-disintegration. The parallelism between the phosphoric-acid and uric-acid elimination which has been present in the cases I have studied seems to be strong confirmatory evidence in favour of this view. In all the cases the food-intake was regulated, and was practically uniform throughout, and the parallel increase could not be attributed to increased ingestion of food. It is well known that nuclein is a phosphorus-containing compound, and the common source of phosphoric acid and uric acid from nuclein-destruction is best illustrated by the following scheme :—

$$\text{Nuclein}\begin{cases}\text{Albumin}\\ \text{Nucleinic acid}\begin{cases}\text{Phosphoric acid}\\ \text{Mother-substance}\begin{cases}\text{Alloxuric, xanthin-, or nuclein-bases.}\\ \text{Uric acid.}\end{cases}\end{cases}\end{cases}$$

The relative amount of uric acid or xanthin-bases produced depends upon the degree of oxidation taking place ; the greater the oxidation the greater the quantity of uric acid produced.

During the acute arthritic manifestations, all the patients had moderate fever, and the objection might be and has been raised that the increased phosphoric-acid excretion is due to increased tissue-combustion resulting from the fever. This point has been kept in view, and the nitrogen-excretion was followed in Cases II. and III. here reported, in order to see whether there was a rise in the total nitrogen-excretion parallel with the uric-acid and phosphoric-acid elimination. While the total nitrogen was moderately increased during the acute attack there was no proportionate increase. An examination of Table III., Case III., will show that during the quiescent period the ratio of uric-acid nitrogen to total nitrogen was as low as 1 to 112 on April 12, whereas on April 23 the ratio had increased to 1 to 57, showing that there was a much greater relative increase in the uric acid than in the total nitrogen-excretion. As the phosphoric-acid curve was practically parallel with the uric-acid curve, although a trifle delayed, it would seem improbable that the increase in the phosphoric-acid output was dependent upon general tissue-disintegration, but rather upon certain tissue-elements, probably the nuclei of the glandular tissues and leucocytes.

The literature contains but scant reference to the association between phosphoric-acid and uric-acid elimination in gout. Bain,[2] in 1899, published a case with charts, in which he came to the conclusion that there was a parallelism in the uric-acid and phosphoric-acid elimination in gout, and believed that nuclein-disintegration was the source of both. Chalmers Watson [3] expresses doubt regarding this possible association.

Not only in the three cases here reported, but also in a considerable number of others studied, the uric-acid elimination has been low in the quiescent intervals, that is, about 0·4 gram daily, and usually much lower. During the acute symptoms the quantity increases and reaches within normal limits, and, according to some authorities, may be above normal, although this has not been the case in the patients whom I have studied. It would hardly seem that the condition of the kidneys could be responsible for this difference. In the three cases here reported, the kidneys all showed evidence of the existence of a chronic interstitial nephritis. The specific gravity of the urine was very low in two, and normal or increased in one, and all showed small traces of albumen and moderate numbers of hyaline and finely-granular casts. The view that there is a retention of uric acid in gout, due to disease of the kidneys, seems somewhat improbable. For why should the kidneys be capable of excreting more uric acid during the acute exacerbations, when their functional activity would be expected to be lowered, owing to the co-existent fever? The same argument, while not disproving the theory, might be employed against the view that uric acid is formed in the kidneys by the union of products brought to them by the renal arteries.

The apparent relationship between the excretion of phosphoric and uric acids in gout, and their relative increase during the acute arthritic manifestations, together with the fact that they are both products of nuclein-destruction, suggest the possibility that in an acute attack of gout there is a marked increase in the quadriurates circulating in the blood, dependent upon a sudden disintegration of the cells of the body that are rich in nuclein. These quadriurates, circulating in a medium rich in sodium carbonate, become converted into the insoluble acid urates, which are deposited in those situations where the temperature is lowest, the circulation poorest, and the percentage of sodium chloride highest, namely, in the cartilages and synovial membranes. It must be admitted, however, that the increase in the phosphoric-acid and uric-acid elimination does not begin until a day or two after the onset of the arthritic pains. The course of the leucocytes was not followed in the cases here recorded.

The purpose of these notes is mainly to draw attention to the apparent association between the uric-acid and phosphoric-acid output in gout. The total nitrogen was estimated by the Kjeldahl method. The uric acid was determined in Case I. by the original Gowland Hopkins process, and in Cases II. and III. by Folin's modification of the Hopkins method. The phosphoric acid was estimated by titration with uranium nitrate. The diets were regulated so as to make them as constant as possible. When changes were made these are noted in the tables. Very brief notes of the cases, with tables and charts, follow:—

Case I.—(General Hospital No. 21,810.) Chronic gout of four years' standing. Tophi in both ears and a very large one over the left tendo Achillis.

A. U., male, aged 41, American, cigar-maker, was admitted to the Johns Hopkins Hospital on January 14, 1898, complaining of swelling and pain in the right knee- and ankle-joints. A father and sister had been "rheumatic," but there was no definite history of gout in the family. The only ætiological factors in the patient's own history were that he had been a hearty eater and a rather heavy beer-drinker. His first attack of arthritis had occurred about four years before admission, the right big-toe and ankle-joints being involved.

CASE I.—TABLE I.

Date.	Urine in cc.	Sp.Gr.	Uric Acid.	Phosphoric Acid.	Remarks.
I. 25–26	1100	1014	0·4301	—	Acute arthritis subsiding. 5th day.
I. 26–27	680	1017	0·3202	—	
I. 27–28	—	—	—	—	Urine accidentally lost.
I. 28-29	350	1019	0·2814	—	
I. 29-30	500	1024	0·3630	—	
I. 30–31	920	1016	0·3661	0·9950	
I. 31– 1	2220	1009	0·2390	0·9440	
II. 1– 2	870	1009	0·0269	0·5410	Feeling well. No acute symptoms.
II. 2– 3	1560	1008	0·1489	—	
II 3– 4	2110	10c8	0·1297	—	
II. 4– 5	2320	1005	0·0893	0·7420	
II. 5– 6	1630	1004	0·0210	0·2450	Acute arthritis in right knee began to-day. Slight fever lasting eight days. Highest 101°.
II. 6– 7	1490	1010	0·0266	0·2730	
II. 7– 8	1390	1008	—	0·8790	
II. 8– 9	1780	1011	0·3867	1·8230	
II 9–10	1660	1012	0·3984	1·4200	
II. 10–11	1860	1013	0·5487	2·1920	Acute symptoms persist.
II. 11-12	1720	1013	0·3887	1·7730	
II. 12–13	2260	1011	0·3921	—	
II. 13–14	1620	1010	0·4829	1·5860	
II. 14–15	2330	1007	0·4729	1·4780	Symptoms subsiding.
II. 15–16	1720	1009	0·3921	1·3990	
II. 16–17	1320	1007	0·2310	0·9870	
II. 17–18	1960	1011	0·1783	0·9900	
II. 18–19	1030	1013	0·0906	0·8230	
II. 19–20	540	1012	0·0729	0·5320	
II. 20–21	440	1011	0·0774	0·4430	Patient feeling well. Out of bed.
II. 21–22	1380	1010	0·0683	0·9770	
II. 22–23	2230	1008	—	0·6430	Uric acid too scanty to estimate. Practically none excreted.
II. 23–24	1680	1008	—	—	Do. do.
II. 24–25	1520	1008	0·1254	0·4400	Acute attack in right tarsal and knee joints began to-day.
II. 25–26	2660	1007	—	0·5320	Practically no uric-acid excretion.
II. 26–27	2430	1010	0·2077	0·9490	
II. 27–28	1630	1010	0·2216	0·7460	
II. 28– 1	1720	1010	0·2365	0·4550	

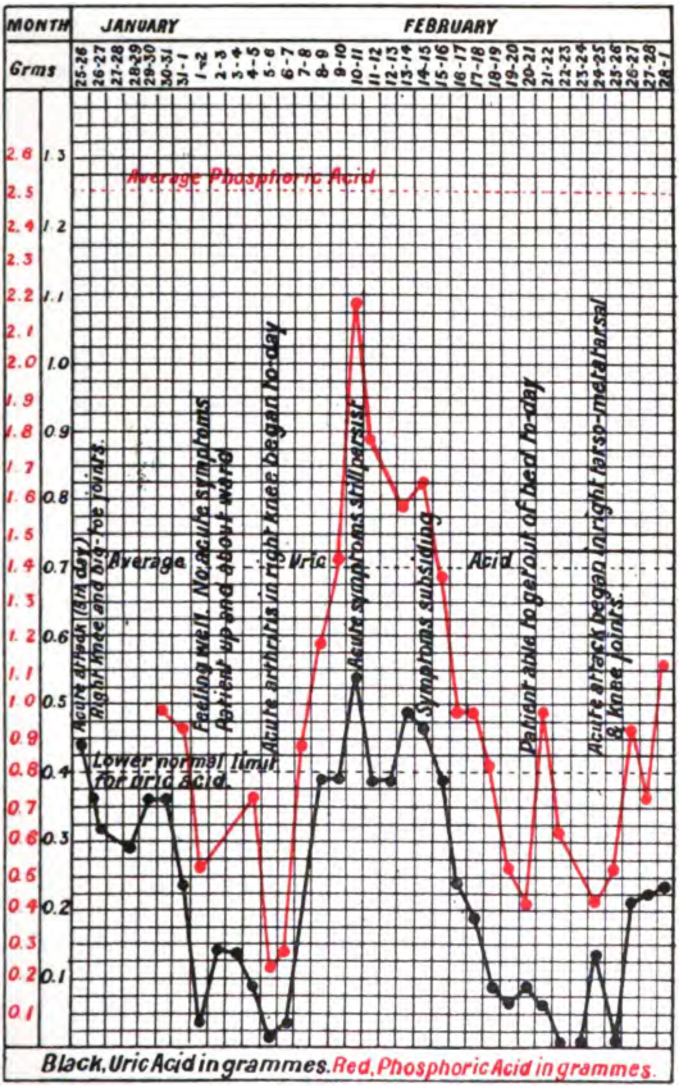
He had suffered from several subsequent attacks, various joints of the body being affected. His last attack began on January 21, four days before admission, and involved the right knee- and big-toe joints. This attack soon subsided, and he was feeling comparatively well when another acute attack, involving the right knee, began on February 5th. His urine was studied during these two attacks, and during the early stages of a third, which commenced on February 24th. The patient was on a constant diet throughout, the details of which will not be given here.

Table I. and Chart I. give the uric-acid and phosphoric-acid excretions in reference to the acute attacks and the quiescent intervals. They require no special comment other than to draw attention to the extremely low uric-acid output in the intervals. The case illustrates, probably better than either of the other two, the close relationship between the two curves.

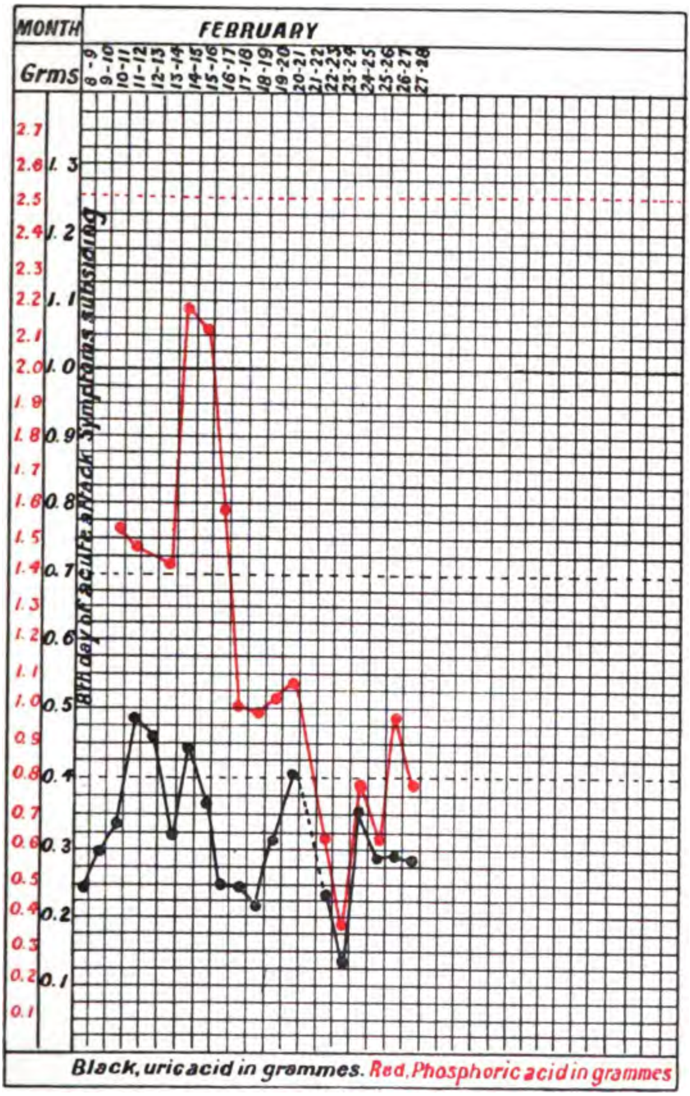
Case II.—(General Hospital No. 21,965.) Chronic gout of seven years' standing. Admitted during an acute attack. Tophi in the right ear.

J. B. H., male, aged 54, American, boiler-maker, was admitted to the Johns Hopkins Hospital February 8, 1898, complaining of swelling of the right knee- and ankle-joints. Excessive indulgence in beer and possible chronic lead-poisoning were the main ætiological factors. His first attack of gout was in the left big-toe joint seven years before admission. He had suffered from several subsequent attacks, in which other joints were also involved. The attack for which he was admitted to the hospital began seven days before entrance with pain and swelling of the right knee, ankle, and tarsal joints. On admission these joints were moderately swollen and stiff, but not painful. The acute symptoms had considerably subsided and patient rapidly improved after admission. There were tophi in the right ear. The patient was placed on a constant diet, and his urine followed from February 8 to February 27 inclusive. There was no return of the acute manifestations during this period. Table II. and Chart II. show the course of the uric-acid and total nitrogen-excretion.

This patient was admitted after the most acute symptoms had begun to subside, and there was at no time a very great relative increase in the uric acid. The largest quantity excreted



CASE I. CHART I.



CASE 2. CHART 2.

was during the twenty-four hours from February 11 to 12. All acute symptoms had subsided by February 15, after which the uric-acid output was decidedly low. The phosphoric acid followed in a general way the uric-acid line, although in the early part of the period of observation the parallelism was not as accurate as later.

CASE II.—TABLE II.

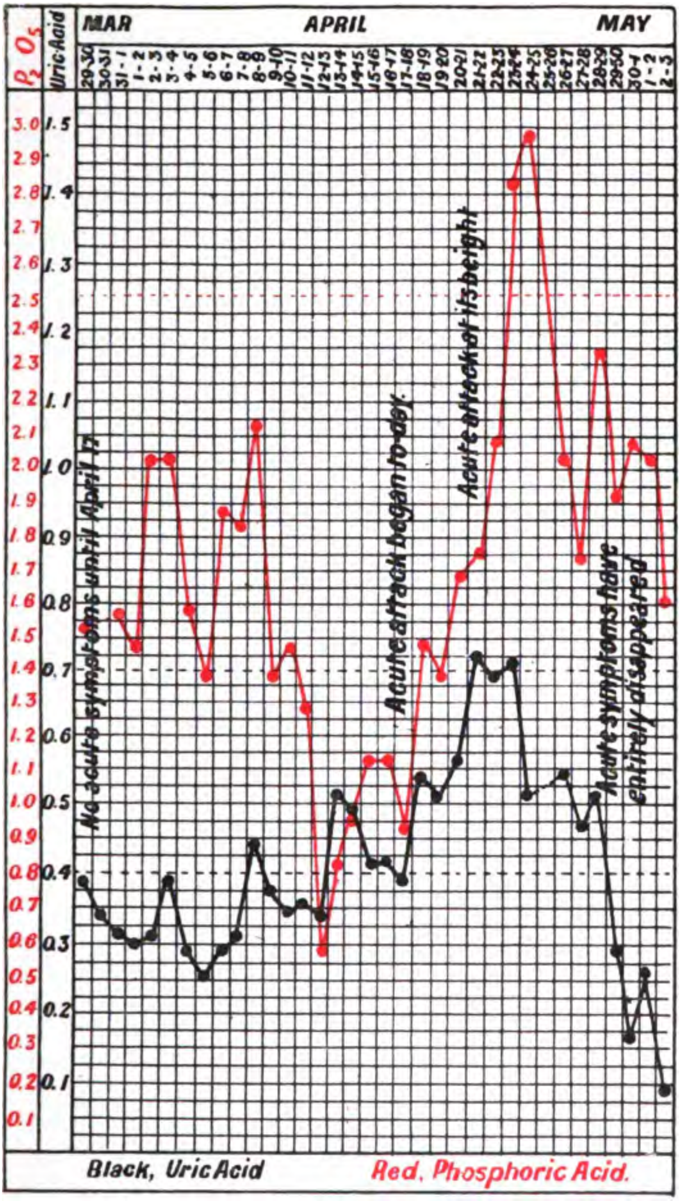
Date.	Urine in c.c.	Sp. Gr.	Total Nitrogen.	Uric Acid.	Uric-Acid N. to Total N.	Phosphoric Acid.	Remarks.
II. 8- 9	1500	1016	—	0·2475	—	—	Urine for 16 hours only. Eighth day of acute attack. Symptoms subsiding.
II. 9-10	1740	1013	10·3704	0·2975	1 : 104	—	
II. 10-11	1010	1017	7·1912	0·3353	1 : 64	1·5150	
II. 11-12	1240	1020	12·6232	0·4811	1 : 78	1·4800	
II. 12-13	1370	1017	11·7272	0·4699	1 : 72	—	
II. 13 14	720	1020	8·2368	0·3240	1 : 76	1·4400	
II. 14-15	1370	1015	12·6588	0·4401	1 : 86	2·1859	
II. 15-16	1010	1017	11·3120	0·3656	1 : 92	2·1009	
II. 16-17	800	1018	8·5280	0·2480	1 : 103	1·5760	
II. 17-18	460	1023	5·9524	0·2442	1 : 73	1·0350	
II. 18-19	300	1036	5·3760	0·2107	1 : 76	0·9900	
II. 19-20	430	1029	7·2412	0·3014	1 : 72	1·0234	
II. 20-21	750	1026	10·7250	0·4192	1 : 76	1·0763	
II. 21-22	—	—	—	—	—	—	Urine lost.
II. 22-23	840	1018	7·4256	0·2385	1 : 93	0·6300	
II. 23-24	720	1013	4·7376	0·1389	1 : 160	0·3024	
II. 24-25	1050	1021	8·2320	c·3643	1 : 67	0·7770	
II. 25-26	740	1022	7·0152	0·2812	1 : 74	0·6031	
II. 26-27	1110	1019	8·2362	0·2897	1 : 85	0·9990	
II. 27-28	1050	1017	6·7200	0·2866	1 : 70	0·7770	

Case III.—(General Hospital No. 38,302.) Chronic gout of ten years' standing. Case considered one of rheumatism at first. Tophi in the ears and in the subcutaneous tissue over the olecranon processes.

J. A. Q., male, aged 47, German, blacksmith, was admitted to the Johns Hopkins Hospital for the fourth time on March 22, 1902, complaining of pain in the hands, elbows, and shoulders. The patient had been a hearty eater and a heavy beer-drinker. These were the only attributable ætiological factors. His arthritic history dated from ten years before. He was admitted first in April, 1895, with polyarthritis which was considered rheumatic in origin. During this admission he had an attack of salicylic-acid delirium. In 1898 he was admitted a second time, and a diagnosis of chronic gout was made. While in the hospital he developed a parotitis, which was not considered to be mumps. On his third admission in January, 1900, tophi were present in the subcutaneous tissue about the elbow-joints, in which the characteristic deposits of sodium biurate were found in histological sections.

The acute attack for which he was admitted the last time, during which the analyses below were made, had lasted about eight weeks. The joints of both upper extremities were involved. He was admitted with considerable pain and swelling of these joints, associated with considerable fever, the temperature reaching 104·3° F. on the day of admission. By March 31 the articular symptoms had entirely subsided, and he was quite comfortable until April 17, when another acute attack began in the right big-toe and knee-joints. The pain and acute symptoms were most severe on April 21, after which they gradually subsided. Table III. and Chart III. show the total-nitrogen, uric-acid, and phosphoric-acid excretion in the urine of this patient.

The analyses in this case were started on March 29 after the acute manifestations had practically subsided. It will be seen from either the table or chart that the uric-acid excretion was a little below 0·4 gram, the lower limit for normal. Then on April 17 an acute attack developed, which was followed in a day or two by a steady rise in the uric acid, until 0·7230 gram was excreted on April 21. With the subsidence of the acute manifestations the uric acid fell, until on May 2 less than 0·2 gram was excreted. The phosphoric-acid curve requires some comment in this case. During the acute attack there was a rise of temperature which corresponded fairly accurately with the rise in the uric-acid curve. There was, however, an unexplainable drop in the phosphoric-acid curve between April 8 and 17, when it failed to correspond at all with the uric-acid curve. A point of interest in this case is the low ratio of uric-acid nitrogen to total nitrogen during



CASE 3. CHART 3.

CASE III.—TABLE III.

Date.	Urine.	Sp. Gr.	Total Nitrogen.	Uric Acid.	Uric Acid N.	Uric Acid N. to Total N.	P_2O_5.	Remarks.
III. 29-30	1760	1008	10·5952	0·3819	0·1273	1 : 83	1·5312	Acute manifestations have subsided.
III. 30-31	860	1017	9·2708	0·3397	0·1132	1 : 81	—	No acute symptoms until April 17.
III. 30- 1	820	1018	8·8068	0·3132	0·1044	1 : 84	1·5580	
IV. 1- 2	1300	1014	10·5560	0·3016	0·1005	1 : 105	1·4560	
IV. 2- 3	2080	1008	11·4816	0·3120	0·1040	1 : 110	2·0259	
IV. 3- 4	1370	1013	11·0148	0·3849	0·1283	1 : 85	2·0687	
IV. 4- 5	1140	1014	10·3284	0·2907	0·0969	1 : 106	1·5960	
IV. 5- 6	1460	1011	9·7528	0·2511	0·0837	1 : 116	1·3870	
IV. 6- 7	1270	1014	10·5768	0·2946	0·0982	1 : 107	1·8542	
IV. 7- 8	1290	1014	11·0166	0·3048	0·1016	1 : 108	1·8369	
IV. 8- 9	2050	1013	14·6780	0·4325	0·1441	1 : 101	2·1443	
IV. 9-10	1950	1011	12·4800	0·3724	0·1241	1 : 100	1·3913	
IV. 10-11	1900	1011	12·7680	0·3477	0·1159	1 : 110	1·4630	
IV. 11-12	1300	1015	11·3620	0·3549	0·1183	1 : 96	1·2870	
IV. 12-13	1330	1017	12·3390	0·3285	0·1095	1 : 112	0·5719	
IV. 13-14	1865	1012	15·1438	0·5166	0·1722	1 : 87	0·8325	
IV. 14-15	2035	1013	14·8555	0·4985	0·1661	1 : 89	0·9645	
IV. 15-16	1550	1014	11·7180	0·4154	0·1384	1 : 84	1·1160	
IV. 16-17	1800	1013	13·0320	0·4176	0·1392	1 : 93	1·1700	
IV. 17-18	1585	1014	11·1591	0·3883	0·1294	1 : 77	0·9020	Acute attack began to-day.
IV. 18-19	2220	1013	14·1192	0·5283	0·1761	1 : 80	1·4740	
IV. 19-20	2220	1012	14·6076	0·5150	0·1701	1 : 85	1·3630	
IV. 20-21	2000	1013	13·9200	0·5632	0·1877	1 : 74	1·6808	
IV. 21-22	3000	1010	16·3800	0·7230	0·2401	1 : 67	1·7520	Acute attack at its height. Began to subside from this time onwards.
IV. 22-23	2510	1010	14·4674	0·6902	0·2300	1 : 62	2·0833	
IV. 23-24	1840	1011	13·6528	0·7139	0·2379	1 : 57	2·8336	
IV. 24-25	1220	1014	12·8100	0·5124	0·1708	1 : 74	2·9890	
IV. 25-26	—	—	—	—	—	—	—	Urine lost.
IV. 26-27	1620	1011	16·4430	0·5362	0·1787	1 : 92	2·0412	
IV. 27-28	1020	1015	10·4448	0·4661	0·1553	1 : 67	1·7380	
IV. 28-29	2140	1009	16·1804	0·5007	0·1669	1 : 96	2·3326	
IV. 29-30	1600	1009	12·0960	0·2928	0·0976	1 : 123	1·9040	No acute symptoms.
IV. 30- 1	940	1013	8·7420	0·1673	0·0557	1 : 156	2·0774	
V. 1- 2	1120	1012	10·5160	0·2553	0·0851	1 : 123	2·0428	
V. 2 -3	1000	1008	7·0000	0·0840	0·0280	1 : 250	1·6000	

the quiescent period, when it reached as low as 1 to 250 on May 2, and the high ratio during the acute attack being 1 to 57 on April 23.

SUMMARY.

The analyses in the cases reported above suggest very strongly a close parallel relationship between the uric-acid and phosphoric-acid excretion in gout. They further point in favour of the view that both are products of nuclein-disintegration. In the quiescent intervals both phosphoric acid and uric acid are markedly reduced below normal. Two or three days after the acute arthritic symptoms commence the phosphoric acid and uric acid gradually increase until they reach the average normal output, or even the upper limit for normal. As the acute manifestations subside both steadily fall and remain below the lower limit for normal until the onset of the next acute attack.

BIBLIOGRAPHY.

[1] Osler: *Principles and Practice of Medicine*, third edition, 1898, p. 412.
[2] Bain: *British Med. Journal*, 1893, II., pp. 1164–1167.
[3] Chalmers Watson: *Encyclopædia Medica*, Vol. IV., p. 221.

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AN ENQUIRY INTO THE CAUSATION OF TUBERCULOSIS IN CHILDREN.*

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FOR the purpose of studying the pathology and ætiology of tuberculosis in children it is convenient to consider the varieties of the disease under three groups, depending upon the distribution of the lesions in the body, viz , pulmonary, alimentary, and general.

Pulmonary tuberculosis, caused by air infected with B. tuberculosis (probably always of human origin), expresses that form of the disease which involves, either as point of entry or as seat of tubercle, any or all of those parts of the body comprised by the term "respiratory system"—air-passages, lungs, and the associated lymphatic glands—and is strictly limited to these alone, there being no evidence of tubercle in the alimentary system. This group includes a number of varieties depending on the extent of the disease, or on its probable point of entry; but, with the exception of certain cases of pharyngeal origin, early or localised disease of the cervical glands, middle-ear disease, &c., they are all characterised by the presence of disease in the lymphatic glands at the root of the lungs. In some cases it is limited to one or two of these glands, there being no obvious sign of tubercular disease in any other part of the respiratory system. These cases have given rise to much difference of opinion, various observers respectively believing that, when the bronchial glands are affected without obvious disease elsewhere, the infection has been carried by the lymphatics from (*a*) the pharynx—tonsils and cervical glands, (*b*) the mucous membrane of the œsophagus, (*c*) the mesenteric glands, (*d*) the thoracic duct, (*e*) by the blood-stream.

Assuming the correctness of any one of these beliefs, it is evident that pulmonary tuberculosis starting in the bronchial glands, and extending to the lungs and other parts of the respiratory system, could not be truly considered to have its origin in every case from infected air, but might equally arise from infected food, &c., and would not therefore necessarily be caused by B. tuberculosis hominis. But it appears probable that these beliefs cannot be justified, and are theoretically unnecessary; moreover, it has been experimentally demonstrated[23] that tubercle-bacilli may penetrate the mucous membrane of the respiratory passages without first infecting the lungs, and this is accepted as a very possible explanation of "primary" infection of bronchial glands. Prof. Sims Woodhead,[22] referring to the caseation of the bronchial glands before the lung itself is affected, writes: "In some cases, no doubt, this is due to a process similar to that described as occurring in the mesenteric glands, where it is accompanied by no permanent lesion of the intestine." Indeed, it seems easy to understand that this might occur frequently, when one considers the pigmented condition of the bronchial glands in healthy persons, even young children, who dwell in towns; for the bacilli or pigment-particles may pass through the healthy mucous membrane to be arrested in the glandular "filters," where they may remain without further result for variable and uncertain periods, depending on the condition of the gland or on the virulence of the irritating particle.

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That the glands at the root of the lung may be infected from primary disease of the pharynx, by extension downwards from the cervical glands, as has been shown to occur in the pig,[17] is possible; though, as Dr. Still says,[21] "Such a method of infection, if it ever occur in the child, must be extremely rare." But the idea that there can be primary disease of the bronchial glands derived from the pharynx, tonsils, or cervical glands, without the intermediate glands of the connecting chain also showing evidence of infection, need not be seriously discussed.

Dr. Guthrie,[11] in a paper on the distribution and origin of tuberculosis in children, suggests that, owing to the arrangement of the lymphatics, it is not improbable that bacilli accompanying mucus or food may be carried almost directly through the œsophageal lymphatics into the mediastinal and bronchial glands. But this is surely not a very likely or possible mode of infection. The mucous membrane of the œsophagus is not to be regarded as an absorbing surface or one from which assimilation can take place. This mode of infection may perhaps be possible in the extremely rare cases of tubercular ulceration of the œsophagus; but in the single case in which the present writer has met with œsophageal ulceration, the bronchial and other thoracic glands, and also the lungs, showed no evidence of tubercular disease.

Referring to infection of the bronchial glands by extension upwards from the mesenteric and retroperitoneal glands, Prof. Woodhead[22] writes: "In other cases it seems to be equally beyond question that the specific virus has passed (*a*) from the mesenteric and retroperitoneal glands, and (*b*) from the peritoneal cavity through the central part of the diaphragm and the broad ligament of the lung, or (*c*) by a more or less circuitous route along the lymphatics of the parietal pleura to the root of the lung." Dr. Still cannot confirm this.

But, although it will be admitted that in cases of general tuberculosis, it is quite possible to trace the disease from the alimentary system along the course suggested by Prof. Woodhead, it must be absolutely denied that primary tuberculosis of the bronchial glands can arise in this manner, since it presupposes the existence of alimentary tuberculosis. It cannot be seriously maintained that bacilli, having passed through the intestinal mucous membrane without producing any lesion, could also pass through the mesenteric glands and the complete series of lymphatics upwards through the diaphragm without infecting them.

Regarding infection of the bronchial glands from the thoracic duct, as suggested by Ponfick,[19] the same objection holds; it would presuppose the existence of alimentary tuberculosis.

Primary tuberculosis of the bronchial glands probably never occurs through the blood-stream. Indeed, primary tubercular infection of any part through the blood-stream is a most rare occurrence, being either the result of congenital tubercle, acquired before birth from a tuberculous mother, or of direct inoculation from a wound. Burdon-Sanderson[5] says, "My own early experiments showed me that peribronchial nodules were amongst the most constant and the earliest of the lesions in artificial tuberculosis of guinea-pigs, as produced by subcutaneous inoculation. In this case the virus is unquestionably not inhaled, but brought to the affected organ by the blood stream." But these are cases of wound-infection; and, moreover, it is wiser not to accept unreservedly the view that those glands which show more advanced or extensive disease are necessarily of older infection than those in which the disease appears less advanced. But the fact that general tuberculosis is brought about by the blood-stream is not questioned. Tubercle-bacilli have been demonstrated in the blood; and Prof. Woodhead[32] refers to the fact that several cases are recorded of general tuberculosis occurring in patients suffering from apical phthisis, after a hæmorrhage and an interval for incubation of 17 to 25 days. Further, operation for local tuberculosis of bone has frequently been noted as the cause of a general infection.

From the above considerations it would appear that "primary" tuberculosis of the bronchial glands and all the other less simple varieties of pulmonary tuberculosis are caused by infected air conveyed by the respiratory channels. It is assumed that this infection is due to the human and not to the bovine or any other variety of B. tuberculosis.

Alimentary tuberculosis, caused by food and other swallowed matters infected with tubercle-bacilli, which may be either of the human, bovine, or any other variety, expresses that form of the disease which involves any or all those parts of the body comprised by the term alimentary system—the alimentary tract and its associated glands and lymphatics—and is strictly limited to these alone; *i.e.*, to cases in which no evidence of tubercle exists in the pulmonary system. This group, like the preceding, includes a number of varieties depending on the extent of the disease and on its probable point of entry; and with the possible exception of certain cases traceable to carious teeth, stomatitis, and pharyngeal infection, they are all characterised by the presence of disease in the lymphatic glands situated in the mesenteries. The simplest form of alimentary tuberculosis comprises those cases where the only evidence of tubercular disease is in one or more of these glands, the intestines through which the infection has presumably passed showing no obvious lesion. It is true that some observers[2] have described cases where there have been lesions of the intestinal mucous membrane without apparently any involvement of the lymphatic glands; but pathologists agree that these very rare cases should be regarded with suspicion. Koch[14a] can recall only two cases; and Dr. Coutts[9] writes: "During a twenty-years' experience at a children's hospital, with exceptional opportunities of seeing *post-mortem* examinations in cases of tuberculosis, I have never met with a single instance." As in the case of primary infection of the bronchial glands, so the question of the possibility of primary infection of mesenteric glands has given rise to much difference of opinion; and probably owing to the indefinite terminology and its loose application there has arisen much misunderstanding. That primary tuberculosis of the mesenteric glands may be derived from infection of pharyngeal and cervical glands is not possible, for the same reasons that were given in considering the case of primary infection of the bronchial glands by this course. Similar objections apply to the suggestion that it may be derived from the pulmonary lymphatics, for, as in the case of bronchial-gland infection from the mesenteric glands, it is quite unlikely that bacilli after passing through the mucous membrane of the bronchi or lungs into the bronchial or mediastinal lymphatics without causing lesions, would also pass through the chain of glands and lymphatics, leaving them unscathed, and produce no tubercular changes until they at last arrived at the mesenteric glands. Such a proposition is too improbable to be discussed. And when one considers the fact, now well established,[8, 10] that it is perfectly possible for tubercle-bacilli to infect the mesenteric glands without producing lesions in the mucous membrane of the intestine, it is consistently a correct assumption that primary tuberculosis of the mesenteric glands—alimentary tuberculosis in its simplest form—invariably arises from the tubercle-bacilli contained in the swallowed matters passing along the alimentary tract. These bacilli may be of any or all the varieties of the tubercle-bacillus.

General tuberculosis comprises all the possible combinations of the other two groups. It may arise from either one of them, or independently from both, by extension through the lymphatics, or by the blood-stream. It is that form of tuberculosis most commonly found at autopsies on children dying from the disease. In fact the other two groups are very rarely met with, since it is the striking characteristic of tuberculosis in children to become generalised. And this seems to be especially the case with the pulmonary form, for it is extremely rare to meet with cases of pure pulmonary tuberculosis *post mortem*. The alimentary form, though no doubt also very rare, is more frequently met with. Is it not suggestive that the dose of the poison, so to speak, either in numbers or virulence of bacilli (human), may be stronger in the case of pulmonary infection, thereby favouring a more rapid generalisation of the disease ? Or is it, perhaps, possible that the peristalsis of the intestine may be in some measure a hindrance to the act of infection, and that the comparatively passive state of the mucous surface in pulmonary infection may favour the absorption of a more constant and stronger dose ? Thus, though the purely pulmonary form is more rare than the purely alimentary form, the origin of general tuberculosis from pulmonary infection is probably much more frequent than from alimentary infection. However, as a rule, the origin of general tuberculosis cannot be determined with any positive certainty.

In connection with the extension of primary tuberculosis, either from the pulmonary or alimentary systems, by the blood-stream, reference should be made to certain cases of tubercular disease of bones and joints, and also of meningitis, found at autopsies, associated with either purely pulmonary or purely alimentary tuberculosis. These are not grouped as cases of general tuberculosis, since their primary source is determinable.

It appears from the above that the facilities for infection by the human B. tuberculosis are much greater than for infection by the bovine or other varieties. It is even possible that all the cases of alimentary infection are by the human variety of the bacillus ; so that the question is rightly asked, whether a child is ever infected by the bovine or other varieties of the bacillus contained in the swallowed matters passing through the alimentary tract.

The present research has been undertaken with a view to decide this question by means of bacteriological methods, by isolating and studying the tubercle-bacilli from every case of alimentary tubercle, that is to say, from every case in which it can be determined with certainty that the infection has occurred from swallowed matter, in accordance with the definition of alimentary tuberculosis enunciated above.

The attempts made by many observers, using various methods of classification, to solve this problem by analyses of statistics obtained from death-returns—with or without autopsies—have created a large literature on the subject, but have produced much conflict and confusion of results, for which varying terminologies are in great part responsible. The expression "tabes mesenterica," for example, has been used in very different senses. This is a clinical term given to the symptoms of a disease the pathology of which is doubtful; it does not occur at autopsies. But it is also often used to mean tubercular peritonitis, and also "consumption of the bowels" or abdominal tuberculosis; but most, if not all, of these cases die of general tuberculosis. It would be interesting to have reliable details of the much-quoted statement of the late Sir R. Thorne Thorne, that the deaths from "tabes mesenterica" and "scrofula" (?) averaged 2,550 per million for the years 1871–80. As the figures stand they are quite valueless and dangerously misleading.

It is generally assumed that "tabes mesenterica" is a tubercular affection, possibly a localised alimentary tuberculosis, that fails from want of virulence to become generalised; and it might perhaps be classed with those other forms of localised tubercular disease seen in the cervical glands, and in those reported cases of accidental inoculation of tubercular (bovine?) matter, which result in the condition known as "tuberculosis verrucosa cutis." This last is a purely local disease, probably never becoming generalised, for, although several cases are described as having died from pulmonary phthisis, the evidence is not free from objections, and is not generally accepted by writers on the subject. It has been suggested that these local wound-infections, cervical-gland infection (probably tonsillar), and "tabes mesenterica" are clinical examples of bovine tuberculosis affecting the human subject.

Again, referring to the term "primary intestinal tuberculosis," Koch[14a] stated "that a case of tuberculosis has been caused by food can be assumed with certainty only when the intestine suffers first, *i.e.*, when a so-called primary tuberculosis of the intestine is found." This does not agree with the grouping and definition given above, and is not usually accepted. It would be useful to know the frequency with which Koch has found "alimentary tuberculosis" as above defined. It is quite possible that the ten cases ("primary intestinal tuberculosis") which he mentions as occurring during five years among the children at the Charité Hospital might be increased tenfold. It will be remembered that it was upon the fact of this great rarity of primary intestinal tuberculosis as defined by him, and in spite of the "multitudes" of bovine bacilli swallowed in milk, that Koch based his opinion that bovine tuberculosis was not transferable to man. Lord Lister,[16] writing in reference to this, says, ". . . in the case of a child fed on unboiled milk from a cow with a tuberculous udder in a room infected with tubercle, it might be fairly open to question whether the bacilli of bovine tubercle imbibed at its meals, or those of human tubercle derived indirectly from the inhaled air, were the more numerous in its intestine;" and he adds that Koch's argument might be used with equal force against the possibility of communicating human tubercle to man. Hence Koch's figures are probably low because of the narrow limit of his definition.

It is likely that the same could be said of Biedert,[3] who, among 3,104 autopsies on tuberculous children, observed only 16 cases of "primary tuberculosis of the intestine," and also of the statement by Baginsky[1] that among 933 cases of tuberculosis in children he never found tuberculosis of the intestine without disease of the lungs and bronchial glands. But how many of those 933 cases showed tubercular disease of the mesenteric glands without intestinal ulceration? And of these what proportion also showed no obvious disease in the lungs and bronchial glands?

In reviewing the published statistics of different workers on this subject, with the object of ascertaining the frequency with which they have found primary alimentary tuberculosis, much difficulty is met with owing to the prevalent misconception that a large proportion of cases showing tubercular disease of the intestine or mesenteric glands, or both, has a direct, or any, relation to the proportion of cases that are really of alimentary origin. It is only in a very few reports that details definitely state that out of such-and-such a number of autopsies on cases of tuberculosis in children in so many there was no obvious sign of disease in the pulmonary system. It cannot be too strongly enforced that it is only in those rare cases met with before the infection has become generalised that it is at all possible to fix the primary seat and probable point of entry.

Prof. S. Woodhead,[22] out of 127 cases of tuberculosis, found 14 in which the mesenteric glands were alone infected. But he found these glands affected in 100 instances, and in 43 cases there was ulceration of the intestines. As no mention is made as to whether the pulmonary system was affected in these cases, one assumes that of the 127 cases only 14 (11·03 per cent.) could be with any certainty traced to an alimentary origin.

Dr. Still[21] reports 269 cases of tuberculosis in the series of 769 consecutive necropsies on children under 12 years of age, made at Great Ormond Street Hospital. He considered that the seat of infection was certainly in the intestine in 53, and probably also in 10 others, making a probable total of 63 cases of alimentary origin. He gives no details of the necropsies, and makes no special mention of the mesenteric glands, nor whether in these 63 cases the bronchial glands were apparently free from tubercle; moreover, he bases his determination of the primary channel on the condition of the lymph-glands, believing that the extent and character of the tubercular process in them may be "taken *ceteris paribus* as an index of the duration of the infection in the organs to which these glands correspond," a principle which is not always to be relied upon, and is not unreservedly accepted.

Dr. Shennan[20] collected 355 cases of tuberculosis from the records of autopsies, extending over a period of 16½ years, at the Royal Hospital for Sick Children at Edinburgh. Relying on the reports of others, he was satisfied as to the "primary focus" in 331 cases, and of these 93 were "alimentary tuberculosis." He gives no details of the grounds on which he based his conclusions, but states that 45 cases showed ulceration of the intestine, prior to affection of the glands, and in 12 cases the glandular lesions were older than those of the intestine. It is difficult to see on what evidence such statements as these could be made. In a latter series of cases he found 11 out of 45 were of "abdominal" origin; so that out of his 374 cases 104, or 27·8 per cent., were considered to be alimentary.

Dr. Carr[6] refers to 120 necropsies made at Victoria Hospital, Chelsea. He states that in 20 cases the disease seemed to have begun in the intestines or mesenteric glands. No particulars are given, and his figures are rather difficult to follow. He found caseous bronchial glands in 96 cases, tubercular disease of the lungs in 90, or pulmonary tuberculosis in at least 96 cases; leaving us 24 cases in which there was no pulmonary affection. He states that there were 5 cases where the mesenteric glands alone were affected; but he considers that 20 cases (16·6 per cent.) were of alimentary origin.

Dr. Batten[2] reports 100 cases of tuberculosis at Great Ormond Street. Of these there was mesenteric infection in 63, but 54 of them also showed disease of the thoracic glands; therefore presumably only 9 were really of alimentary origin. He noted four cases of intestinal ulceration without the mesenteric glands being affected. If in these 4 cases the thoracic glands also showed no signs of tubercle, we may conclude there were in all 13 cases (13 per cent.) of primary alimentary infection.

Heller,[12] of Kiel, considered that out of 140 cases of tuberculosis 53 (37·8 per cent.) were of alimentary origin. No details of the necropsies are given, and in view of his extraordinarily high figures their accuracy in the present sense is to be questioned. He compares his results with those of Councilman, Mallory, and Pearce at Boston, U.S.A. These authors report 35 cases, 13 (37·1 per cent.) being regarded as alimentary in origin. Here, again, no details are given.

Colman[7] records 59 cases, and Guthrie,[11] 77; but the figures of these observers are of little value owing to the absence of details necessary for analysis.

Northrupp[18] found in 125 cases that 3 (2·4 per cent.) were of alimentary origin, the remaining 122 cases all showed affection of the bronchial glands.

Kossel[15] gives careful autopsy-notes of 22 cases, from which it appears that only 1 case (4·54 per cent.) is to be regarded as of alimentary origin.

Bovaird[4] records 75 cases from the Foundling Asylum, New York. The bronchial glands were affected in every case.

Emmet Holt[13] found, after careful study of the necropsies of 119 cases of tuberculosis from the Foundling Asylum and the New York Babies' Hospital, that in none was it probable that the primary lesion was in the stomach or intestine, though there was mesenteric disease in 35 per cent., and intestinal ulceration in 37 per cent. of the cases.

Tabulating the above records we get :—

Observer.	Total Number of Cases.	Number of Alimentary Cases.	Per Cent.
Woodhead - -	127	14	11·03
Still - - -	269	63	23·42
Shennan - - -	374	104	27·8
Carr - - -	120	20	16·6
Batten - - -	100	13	13
Heller - - -	140	53	37·8
Councilman - -	35	13	37·1
Northrupp - -	125	3	2·4
Kossel - - -	22	1	4·54
Bovaird - - -	75	0	0
Holt - - -	119	0	0

To this may be added the results of the present research as far as it hath progressed.

Price-Jones - -	21	6	28·5

Comparison of these results is difficult, for they have but little similarity ; but agreement and contrast are at once seen if, after discarding the questionable figures of Heller and Councilman, a table is drawn up in which the records are grouped according to the place at which the observations were made ; thus :—

Observer.	Place.	Total Number of Cases.	Number of Alimentary Cases.	Per Cent.
Still - - -	London -	269	63	23·42
Carr - - -	,, -	120	20	16·6
Batten - -	,, -	100	13	13·0
Price-Jones - -	,, -	21	6	28·5
Total - -	,, -	510	102	20·0

Observer.	Place.	Total Number of Cases.	Number of Alimentary Cases.	Per Cent.
Woodhead - -	Edinburgh	127	14	11·03
Shennan - -	,,	374	104	27·8
Total - -	,,	501	118	23·5
Northrupp - -	New York -	125	3	2·4
Bovaird - -	,, -	75	0	0
Holt - - -	,, -	119	0	0
Total - -	,, -	319	3	0·94
Kossel - -	Berlin -	22	1	4·54

This arrangement shows a striking agreement of the results obtained in London and in Edinburgh, and an equally striking contrast between them and those obtained in New York. But it is very clear that these are but artificial agreements and contrasts, for they combine such different results as those of Woodhead and Shennan, and make them agree with the London group of observations, which again combine such diverging percentages as 13 and 28·5; and, as already stated, the methods by which these figures have been arrived at are most varied and indefinite. Indeed, statistics should not be compared unless they are collected by identical methods, with strict adherence to a well-defined plan of classification. So far, then, statistics on the ratio of alimentary to other forms of tuberculosis must be held to afford but little assistance in the investigation of this subject.

Great stress is laid by some observers on the age-incidence of tuberculosis, and roughly it is agreed that there is a maximum of cases of alimentary tuberculosis between the ages of 1 and 5 years. From this it has been eagerly assumed that ingestion of cows' milk containing the B. tuberculosis bovis is the cause of the large death-rate from tuberculosis in children. The steps of the argument being that at this age (*a*) the child is weaned; (*b*) it is fed on cows' milk; (*c*) the milk is loaded with bacilli of bovine tuberculosis. That the majority of children are weaned between the first 9 to 12 months of life is probably true; but the rest of the argument is pure assumption and probably false.

It is intended as a part of this research to ascertain, where possible, whether those children who die of alimentary tuberculosis have been fed on cows' milk, and if so, to endeavour to obtain roughly an approximate estimate of the amount of such milk consumed. For it is certainly open to question whether, after they are weaned, the children of the poor, who represent the patients at a children's hospital, are usually fed on cows' milk. One has been told that half a pint of milk, or even less, suffices for two parents and three or four children, and is used to mix with their tea. Assuming that the milk is loaded with bovine tubercle-bacilli, surely the chances of any one of the children becoming affected by the few drops of milk that have helped to cool down their tea from 90° to 80° C. would not probably be very great. Occasionally also it happens that the milk is boiled. But when it is considered that the number of tubercle-bacilli in milk is usually very small, the chances are lessened; and yet more so if, as is frequently the case, the milk supplied is not fresh cows' milk, but some tinned preparation preferred for its price and sweetness. The same may be said for the chances of infection by the human tubercle-bacillus; for this variety may very possibly occur in even greater numbers in milk as served at table than the bovine variety. So that it does not seem very probable that cows' milk is a frequent cause of tubercular infection in children after they are weaned.

Thus the question becomes limited to bottle-fed infants under one year old. And it cannot be denied that the chances of their becoming infected do really exist, since milk is their only food; and there is little doubt that the bacterial contaminations of milk are responsible for the high death-rate among these infants. But tuberculosis is comparatively rare at this age; and, besides, in those cases met with, it would still be an open question whether the bacilli are not quite as likely to be the human as the bovine variety. Lastly, infants are often fed on artificial foods.

However, the influence of age (1 to 5 years) is important for many and far more important reasons than the ingestion of occasional small quantities of cows' milk. This is the first of a series of revolutionary periods of life, involving the passing from infancy to childhood, the period of dentition, the development of the secretory glands, and the corresponding change in the nature of the food. The lymph-glands of the body having so much extra work thrust upon them, it should not be a matter of surprise that in autopsies on children dying from any cause, not tuberculosis, at this age, we find these glands comparatively enormously enlarged. In fact this period of life is well described as the "lymphatic period." Further, this is the period when the child first comes in contact with other things; its sphere of action is widened; it crawls, and learns to walk; it mixes more with its fellows and the world's dirt—bringing into play those curiously vague and meaningless properties, so often referred to, of "predisposition" and "hereditary tendencies." And, perhaps, yet more forceful, this is the period for acquiring various infectious fevers, among which whooping-cough and especially measles should undoubtedly be underlined as the great predisposing causes to the acquisition of tuberculosis. And, again, a force probably of equal importance lies in the fact that at this period of life few children, particularly the children of the poorer classes, do, or are able to, breathe through their noses. All the impurities of the air are taken into the mouth, some inhaled, some swallowed with the saliva or food. It would be interesting to know what proportion of swabs, taken hap-hazard from the mouths of a troop of children playing in their gutter, would produce tuberculosis in guinea-pigs. This inability to breathe through the nose is of course part of the lymphatic process of this time of life, which involves enlarged tonsils, narrow fauces, and great frequency of large adenoid masses in the nasopharynx, making normal respiration impossible.

The point that I wish to urge is that the source of the disease is probably the polluted air, and not the food at all, unless this also has been exposed to the tubercular conditions of its surroundings. For it cannot be too much insisted upon that the tubercle-bacillus is found in the dust of close, overcrowded rooms; that it arises from the expectoration and excretions of persons suffering from tuberculosis. It is most probably represented by a large group of varieties, some noxious, others harmless for human beings; some, like the acid-fast streptothrices, being readily distinguishable, others, like the avian and bovine varieties, being more closely allied and less easily separated—the bovine variety especially, until recent years, being regarded as identical with the human bacillus.

Before leaving this part of the subject, reference should be made to several much-reported cases of tuberculosis, apparently resulting from the ingestion of milk from tuberculous cows. Ollivier has reported a series of cases occurring in a girls' school, where 13 pupils, within a few years, sickened of tuberculosis, and six cases were fatal. On enquiry it was found that the boarders had been drinking milk from a tuberculous cow. But on further analysis it appears that only one necropsy was made, and as Koch [14b] in criticising these cases goes on to say: "A direct infection from one person to another has not been publicly brought under consideration at all, and yet the course of this little epidemic of tuberculosis would have been exactly the same, if one of the boarders or a pupil had suffered from tuberculosis of the lungs, and infected a number of her companions through the sputum. Something of the kind has often occurred, and its possibility would have to be considered among all the circumstances. But, even apart from this consideration, the alleged classical case vanishes away, and even proves the reverse, as the result of a second communication by Ollivier, which he found himself obliged to make at the next session of the Academy, in consequence of better information. He actually had to declare that he was mistaken; that the milk from the cow in question was not drunk by the boarders, but by the teaching staff and the domestic servants of the establishment. Among those persons who had regularly partaken of the suspected milk not a single case of tuberculosis had occurred."

Again, take the series of cases recorded by Huls as occurring in a miller's family of nine persons, who for years are said to have fed on the milk, butter, and meat of tuberculous animals; seven cases being fatal. Koch [14b] gives the following instructive account and criticism: "First the mother sickened, but recovered again after some months. In the following year the youngest child sickened and died. In the same year a son, aged 18 years, became ill and died. A year later followed a son, aged 23 years. In the next year the mother got ill again and died; then followed a girl of 16, then the father, and lastly, a third son. That, among the relations of the illness to the family here indicated, contiguity, the dwelling together of the sick and healthy, and so the transference from one to the other should be excluded, is to me incredible. Every expert in infectious diseases will be convinced without any further evidence that there was in this case a continuous chain of contact-infections and not a group of illnesses arising from infected food. In order to be able to accept the last as the origin, the illnesses must have ensued in the course of six months or a year at least; they could not, as was the case here, be protracted over a series of years." Koch[14b] also refers to 28 isolated cases reported by Nocard, Stang, Uffelmann, and others, and after careful examination he concludes that "these do not merit any great confidence in and for themselves." But he adds, "I neither can nor will contest the possibility that single cases may occur. But in order to be convincing they must fulfil certain conditions." He then proceeds to state the following postulates:—"(*a*) Certain proof of tubercle in general, and where possible the primary focus must be supplied. In adults we must, therefore, require the existence of unassailable clinical symptoms, and a necropsy when these are not present. In children the clinical symptoms are much too uncertain, and so a necropsy is always requisite with them. (*b*) Other sources of infection must be excluded with certainty. The assurance that the person in question comes of a healthy family is under no circumstances sufficient. There are numerous other possibilities of infection either inside or outside the family which come up for consideration." And he draws attention to the researches of Preisich and Schültz, and of Dieudonné, on the occurrence of tubercle-bacilli round the nails of children. (*c*) "In each case of alleged infection from milk affected with *Perlsucht* the condition of the rest of the people who have taken the same milk should be borne in mind. These fellow-consumers form to a certain extent a control experiment, and if of the numerous people who have drunk the suspected milk only a single one sickens, this weighs decidedly against the belief that this one person was infected by the common food." And he adds, "With enteric, too, if only one of all the people who had drunk the same milk contracted typhoid, we should on this ground alone immediately relinquish the suspicion that it could depend on a milk-infection." It is, however, very unlikely that many authorities will allow the truth of this comparison. There is surely no analogy between tuberculosis and typhoid. (*d*) "The source of the milk should be attended to. Since in recent years it has become more and more evident that milk containing tubercle-bacilli is yielded only by such cows that suffer from tuberculosis of the udders, the general statement that someone has drunk milk from a cow suffering from *Perlsucht* no longer suffices to prove to us that Perlsucht-bacilli have really reached his digestive organs. The milk must be from a cow with tuberculosis of the udder."

Again, from the other point of view of the question, numerous instances have been recorded of persons and families who have consumed milk from tuberculous cows and yet have remained free from disease. Shennan[20] refers to the example of Clifford Allbutt, who allowed his own family and servants, as well as two other families living near, to consume the highly "bacilliferous" milk of a favourite cow; not one individual developing the slightest trace of tuberculosis.

Also Emmet Holt[13] refers to the often-quoted instance occurring in an American city where 45 per cent. of the Jersey cows gave a positive reaction with tuberculin, and supplied milk to many families (mostly wealthy) for over ten years; investigation showed that only one child out of many hundreds fed on the milk developed tuberculosis, and in this case it was by no means established that the milk was the source of infection. Of course this record requires more detailed information as to whether the milk contained tubercle-bacilli; and as to the one case of disease, the seat of infection should have been given. From the above records it would appear very doubtful if tuberculosis in children arises from the ingestion of milk from tuberculous cows.

The material for this research is being obtained from the *post-mortem* examinations made at the East London Hospital for Children, Shadwell, by the kind permission of Dr. Eustace Smith, Dr. Coutts, and Dr. Morley Fletcher, to whom, and also to the pathologist, Dr. Dudgeon, for his invaluable help, the present writer offers his best thanks. The bacteriological part of the work is being carried out at the Gordon Research Laboratory, Guy's Hospital, and the writer wishes to acknowledge the great assistance he has received from Dr. Ainley Walker, who kindly made the inoculation-experiments for him.

The bacteriological portion of the research is yet in a very early stage, the isolation and cultivation of tubercle-bacilli being a very difficult and lengthy process ; and it is hoped to give a complete account of the work in connection with each case of alimentary tuberculosis in a subsequent paper. But as an example of the preceding definitions, and of the method adopted in choosing the cases, it will probably be considered advantageous and interesting to give in this place details of the autopsies of all the cases of tuberculosis so far met with since the commencement of this work.

From February 23 to May 11 55 consecutive *post-mortem* examinations were attended. Of these 21 were cases of tuberculosis; and readers of the following notes will be able to confirm the statement made above, that of these 21 cases 6 were undoubtedly of alimentary origin.

Case 1. February 23.—G. B., 6 years. There were no obvious signs of tubercle in the thorax; the lungs and bronchial glands appeared to be quite healthy. A few enlarged caseous mesenteric glands were on the point of rupture. There was no evidence of tubercular lesions in the intestine; other abdominal glands were not affected; and there were no signs of tubercle in any of the abdominal viscera. A few scattered tubercles were visible on the meninges in the interpeduncular space and Sylvian fissures; a large tubercular caseous mass occupied the substance of the cerebellum, especially the right lobe. There was no evidence of mastoid disease. This case is regarded as having an alimentary origin.

Case 2. February 24.—G. L. S., 2½ years There were no obvious signs of tubercle in the thorax; the lungs and bronchial glands appeared to be quite healthy. There was general peritonitis; the bowels were matted together and with the omentum, which was much thickened and had on its surface and in its substance numerous small and large miliary tubercles. The mesenteric glands were much enlarged, and the iliac glands showed caseation; there was most extensive tubercular disease extending from the œsophagus to the rectum, showing recent ulcerations, old scars, and miliary tubercles. There were tubercles on the capsule of the liver and spleen, and one old calcareous nodule in the splenic substance. There were tubercles on the abdominal surface of the diaphragm, and on its pleural surface; at the attachment of the costal margin there were several enlarged glands forming a chain, the outermost two being the largest. There was no evidence of tubercle on the meninges; the brain appeared healthy; there was no mastoid disease. The cervical glands were healthy. This case is regarded as having an alimentary origin.

Case 3. March 2.—C. M., 7 years. Both lungs showed most extensive tubercular disease; on the epipleura of both sides there were a number of large tubercles on the point of breaking down; and on section the lungs showed enormous numbers of large caseous tubercles. The mediastinal, bronchial, and tracheal glands were enlarged, and many were caseous. The pericardium and heart were healthy, as also were the larynx and trachea. The general and intestinal peritoneum appeared healthy; the mesenteric and portal glands were enlarged and tuberculous, but none were caseous; the liver was enlarged, and both on the surface and throughout its substance there were enormous numbers of small miliary tubercles. The spleen was large (7 ozs.), and massed with caseous tubercles; the kidneys were pale, and showed numerous caseous tubercles at the surface; the meninges appeared healthy, and there was no mastoid disease. This is a case of general tuberculosis, the seat of primary infection being either pulmonary or alimentary, or both.

Case 4. March 3.—E. R., 4 years. There were numerous enlarged tuberculous glands in the anterior mediastinum, and between the pericardium and the upper lobe of the right lung there was a large fibro-caseous mass, the size of a tangerine orange, adherent to both serous sacs; all the glands of the thorax and neck showed a similar caseous condition. Tubercles were scattered over the surfaces of both lungs, and in the upper part of the lower lobe of the right side there was caseous consolidation. The mesenteric glands and intestines showed no signs of tubercular disease; but the portal glands were tuberculous, and there were miliary tubercles in the liver, spleen, and kidneys. There were a few tubercles on the meninges about the interpeduncular space, the optic commissures, and along the Sylvian fissures, and a few on the left occipital lobe and under surface of the cerebellum. The left middle ear contained a little pus. This case is regarded as one of general tuberculosis, the primary seat of infection being probably pulmonary, and possibly from the middle ear.

Case 5. March 5.—A. T., 10 months. The right lung was full of miliary tubercle; the upper part of the left lung was adherent to the chest-wall, enclosing a sacful of fluid; there was no pleurisy on the right side. Most of the thoracic glands were enlarged and caseous; there were miliary tubercles on the pericardium; there was caseation of the mesenteric glands, but no signs of tubercular disease in the intestines. There were a few recent miliary tubercles in the liver, spleen, and kidneys; the peritoneum appeared healthy. There was much shrinkage on the left side of the brain, which was very soft and broken down in parts; there was complete thrombosis of the left middle cerebral artery; adherent with the external wall along the Sylvian fissure were patches of thick lymph containing a few miliary tubercles. The right side of the brain appeared healthy. There was no sign of mastoid disease. This is regarded as a case of general tuberculosis, the primary seat of infection being probably pulmonary.

Case 6. March 6.—B. G., 10 months. This was a case of gangrenous varicella. There were no signs of tubercular disease in the thorax; and, excepting some hæmorrhages into the visceral surface of the pleura on the left side, the thoracic viscera and glands appeared healthy. There were large transverse tubercular ulcerations of the intestine at the lower end of the ileum and commencement of the big gut; the mesenteric glands were not much enlarged, but some showed recent tubercles, and a few were caseous. There was nutmeg liver, and the spleen was large, but no tubercles were noticed in these organs. There was old morbus cordis; the other viscera seemed healthy. There was no sign of disease in the meninges, brain, or mastoid. This is regarded as a case of alimentary tuberculosis.

Case 7. March 11.—M. W., 2 years 3 months. Both lungs were adherent to the chest-wall by dense attachments; the left lung was congested and showed numerous miliary tubercles, also a few small tubercular masses about the size of a pea; the right lung was consolidated, but there were no tubercles to be seen. The bronchial and tracheal glands were large and fibro-caseous. There was no evidence whatever of tubercular disease in the abdominal cavity. The brain appeared healthy. This is regarded as a case of pulmonary tuberculosis.

Case 8. March 13.—L. K., 2 years. There were enormous numbers of quite recent tubercles in both lungs; recent pleuritic effusion; the bronchial and mediastinal glands were enlarged and caseous; the heart was healthy. There was general miliary tubercular peritonitis; a large number of ulcerations of the intestine; the mesenteric glands were enlarged and caseous; miliary tubercles were scattered on the surfaces of the liver, spleen, and kidneys, and also on the meninges about the Sylvian fissures, under surface of the cerebellum and interpeduncular space. This is regarded as a case of general tuberculosis, and the primary seat of infection is either pulmonary, alimentary, or both.

Case 9. March 18.—G. F., 10 months. There was bronchopneumonia, but no signs of tubercle in the thorax, the bronchial glands appearing to be healthy; several of the mesenteric glands were enlarged and over-hard on section, but showed no signs of caseation and did not seem to suggest tubercle; but an emulsion made from them produced tuberculosis in two guinea-pigs subcutaneously inoculated. The liver was enlarged and fatty, but neither here nor in any of the other viscera was there any obvious sign of tubercle. It may be here noted that this case was reported to be entirely breast-fed. (Alimentary tuberculosis.)

Case 10. March 18.—W. I. F., 3½ years. There was no sign of tubercle in the thorax, the viscera and glands appearing healthy; there was most extensive tubercular peritonitis; much matting together of the intestines, though they were not adherent to the abdominal wall. There were enormous numbers of tubercular ulcerations in the ileum and large gut; they had raised edges and firm sloughy bases, were about two inches in diameter, and in places completely encircled the gut. There were enormous numbers of enlarged and caseous mesenteric glands; tubercles were noted on the liver, but not on the other viscera; there was no meningitis nor mastoid disease. (Alimentary tuberculosis.)

Case 11. March 21.—G. B., 2 years. There was recent pleural adhesion on both sides; the left lung showed consolidation of the upper lobe, and on section exhibited patches of caseating tuberculous deposits, and collapse of the lower lobe. On the right lung there were many scattered tubercles, and early caseation at the anterior margin of the upper lobe; the bronchial glands were enlarged and tuberculous. There were tubercles on the peritoneum, on the capsule of the liver and spleen; intestinal ulceration; caseous mesenteric glands—two large caseous glands pressing on the bile-duct; there was no meningitis or mastoid disease. (General tuberculosis.)

Case 12. March 20.—E. W., 1⅓ years. There were scattered miliary tubercle on both lungs, but no consolidation; an enlarged fibro-caseous condition of the bronchial and tracheal glands. There were no obvious signs of tubercle in the bowel, and the mesenteric glands appeared healthy; but there were several caseous hepatic glands, and a few scattered tubercles in the spleen; there was no meningitis or mastoid disease. (General tuberculosis.)

Case 13. March 18.—L. W., 8 months. There was miliary tuberculosis of both lungs; enlarged and caseous tracheal and bronchial glands; large, probably old, ulcerations in the intestines; great numbers of caseous mesenteric and hepatic glands; scattered tubercles on the liver, spleen, and kidneys; no meningitis. (General tuberculosis.)

Case 14. March 26.—W. R., 1 year 5 months. There was miliary tuberculosis of both lungs and pleura; an old fibro-caseous condition of the bronchial and mediastinal glands, and extensive intestinal ulceration (probably not very old). A great number of enlarged mesenteric glands were present, some being caseous, but the disease seemed to be of more recent date than that of the bronchial glands. There were miliary tubercles and two small ulcerations on the mucous membrane of the bladder; also in the left ureter and kidney. There was acute miliary basal meningitis about the Sylvian fissures, interpeduncular space, and under surface of the cerebullum; but no tubercles were noted in the ventricles; there was no sign of mastoid disease. (General tuberculosis.)

Case 15. March 30.—F. H., 8 years. There was miliary tuberculosis of both lungs, and consolidation of the upper lobe at the apex; old caseous fibrosis of the bronchial glands, scattered tubercles on the pericardium and heart, but no adhesions; tubercular peritonitis, the intestines being matted together and to the omentum, and the peritoneal cavity being obliterated; enormous numbers of tubercles covered all the viscera, all of which, except the spleen, showed tubercles in their substance. The liver showed tubercular cavities; there was tubercular ulceration of the intestine, and very large numbers of the mesenteric glands were caseous. There was extensive meningitis, both basal and cortical, especially over the left motor area, where it penetrated into the white matter; there was a large deposit of caseous material along the longitudinal fissure on the left side, in the region of the Rolandic area; the lateral and the third and fourth ventricles were dilated, containing an excess of turbid fluid. There was no evidence of mastoid disease. This patient was admitted for right-sided hemiplegia, and a diagnosis of cerebral tumour had been made. (General tuberculosis.)

Case 16. April 24.—L. S., 2½ years. The lungs, bronchial and mediastinal glands seemed to be healthy, there being no sign of tubercle in any part of the pulmonary system. The abdomen was much distended, especially below the umbilicus, and a most extreme state of tubercular peritonitis was present; the abdominal cavity being quite obliterated, and the intestines all matted together and to the other viscera, and adherent to the abdominal wall. They were bound down by thick infiltrated bands of omentum and mesentery, and the examination of the abdominal viscera was accordingly made very difficult; the bowel readily ruptured in the neighbourhood of the cæcum, where there was a large ulcerated surface on the mucous membrane about three inches in widest diameter. There were numerous large caseous mesenteric glands, and caseous deposits on the liver and spleen. The diaphragm was adherent to the upper surface of the liver; on the pleural surface of the diaphragm were large caseous glands similar to those noted in Case 2; and on the right side, apparently in continuity with the disease of the liver, there was a small patch of tubercular diaphragmatic pleurisy. The brain was not examined. (Alimentary tuberculosis.)

Case 17. April 27.—C. B., 1⅓ years. There were much enlarged and caseous glands on the neck, especially on the left side; two large shaggy-walled cavities on the upper lobe of the left lung, the left pleural cavity being obliterated; miliary tubercles in both lungs, with large caseous mediastinal and bronchial glands especially lying in the septum between the upper and the lower lobes. There were caseous masses on the upper surface of the diaphragm, and on its under surface many smaller tuberculous deposits. Similar deposits were found in all the abdominal viscera, including the suprarenals and kidneys. There was a tubercular ulcer on the mucous surface of the stomach; ulcerations of the intestine; masses of large and caseous mesenteric and other abdominal glands; but no sign of peritonitis, excepting a few recent tubercles on the peritoneal surface of the gut. There was extensive disease of the left mastoid bone, tubercular meningitis, and softening of the cerebral substance. Over the left occipital cortex in the median line were numerous miliary tubercles, and a small caseous patch in the frontal region in the white matter. The right mastoid was healthy. (General tuberculosis, the primary seat being most probably in the mastoid.)

Case 18. April 30.—C. H., 3 years. Old and recent tubercles were present in both lungs; all the thoracic glands were enormously enlarged and caseous; there was tubercle on the surface of the heart. There was extensive tubercular peritonitis, the upper part of the abdominal cavity being obliterated; the liver and spleen were full of tuberculous masses; there was tubercular ulceration of the colon near the cæcum; the mesenteric and other abdominal glands were enlarged and caseous; there were tubercles in both kidneys. Tubercular meningitis was found, but no tubercle in the brain-substance; the mastoids healthy. (General tuberculosis.)

Case 19. May 4.—J. B., 13 months. Empyema, pyopericarditis (from the pus was obtained a pure culture of pneumococcus). There were tubercular cavities and miliary tubercles in the right lung, no lesions in the left lung, many caseous bronchial glands. There was an old tubercular nodule and a few miliary tubercles in the spleen; but no signs of tubercle in the other abdominal viscera; the mesenteric glands appeared to be healthy. There was no meningitis, nor was there mastoid disease. (General tuberculosis, probably primary pulmonary.)

Case 20. May 4.—R. S., 6 months. Miliary tubercle was found and also extreme tubercular consolidation of both lungs, which were undergoing caseation; no cavity-formation; the bronchial glands were much enlarged and caseous; the pleuræ were healthy. There were a few caseous mesenteric glands, but no ulceration of the intestines; no tubercles were noted on the peritoneum, but two caseous pin-head deposits were seen on the liver and on the spleen. There was no meningitis; the mastoids appeared to be healthy. (General tuberculosis, probably primary pulmonary.)

Case 21. May 7.—C. O., 1 year 10 months. There were caseating cervical glands with tubercular bronchopneumonia of both lungs and scattered tubercles on the pleuræ; the mediastinal and bronchial glands were caseous; at one place at the root of the left lung they had broken down and formed a cavity into the lung; large tuberculous deposits on both surfaces of the diaphragm. Caseous mesenteric and portal glands were found; the mesenteric being especially enlarged in the region of the last 10 inches of the ileum. There was large and probably recent tubercular ulceration of the bowel showing tubercles on the peritoneal coat of the ulcers. There were scattered tubercles on the liver, spleen, and kidneys; and tubercular meningitis, especially about the optic commissure; no mastoid disease. (General tuberculosis.)

The above cases may be tabulated as under:—

Case No.	Age of Child.	Form of Tuberculosis.
1	6 years	Alimentary.
2	2½ ,,	Alimentary.
3	7 ,,	General.
4	4 ,,	General—? mastoid.
5	10 months	General—? pulmonary.
6	1 year 10 months	Alimentary.
7	2 years 3 months	Pulmonary.
8	2 ,,	General.

Case No.	Age of Child.	Form of Tuberculosis.
9	10 months	Alimentary.
10	3½ years	Alimentary.
11	2 ,,	General.
12	1½ ,,	General.
13	8 months	General.
14	1 year 5 months	General.
15	8 years	General.
16	2½ ,,	Alimentary.
17	1½ ,,	General—? mastoid.
18	3 ,,	General.
19	13 months	General—? pulmonary.
20	6 ,,	General—? pulmonary.
21	1 year 10 months	General.

The age-column is not of course of much value, since it is the age at death that has been recorded, and this has no relation to the age at the date of infection. The cases may also be arranged thus:—

Alimentary tuberculosis	6 cases.
Pulmonary tuberculosis	1 case.
Pulmonary, probable, including 2 cases of primary mastoid (?) *	5 cases.
General tuberculosis	9 cases.
Total	21 cases.

From the foregoing statements we learn that in this country alimentary tuberculosis occurs in about 25 per cent. of all the cases of tuberculosis in children; that this is possibly caused by the B. tuberculosis bovis, but that the chances of its being caused by the human or other varieties of the tubercle-bacillus appear to be greater. It thus seems desirable that, where possible, the bacillus causing alimentary tuberculosis should be isolated and studied, so as to determine (*a*) whether this is always the same organism, (*b*) whether it is identical or not with organism of pulmonary phthisis, (*c*) what relationship it bears to the organism found in the milk from a cow with a tuberculous udder, and (*d*) to the other so-called varieties of the tubercle-bacillus; thus enabling one, perhaps, to say whether these different forms of bacilli are genuine varieties with fixed biological characters, or whether they are but false varieties with apparent differences depending not on the organism itself, but on the reaction of the tissues of various hosts.

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* Since in these two cases of mastoid disease no bacteriological examination of the pus was made, it is a possible criticism that these were cases of pneumococcic infection; but this suggestion must be left until the important subject of mixed infections is dealt with, when it may be found that the association of the pneumococcus with the tubercle-bacillus is an all-important factor in the production of tuberculosis in children.

The work is now proceeding, and it is hoped in a future statement to give account of the bacteriological aspects of some of the cases recorded in this introductory paper.

LIST OF REFERENCES.

[1] **Baginsky**: Referred to by Koch [14] (*a*).

[2] **Batten**: "The relative Frequency of Tuberculous Infection of the Lymphatic Glands in Children," *Bart. Hosp. Reports*, 1895, Vol. XXXI., p. 183.

[3] **Biedert**: Referred to by Koch [14] (*a*).

[4] **Bovaird**: (*a*) *Archives Pediat.*, 1901, Vol. XVIII., No. 12; (*b*) *New York Med. Journ.*, 1899, Vol. II., p. 1.

[5] **Burdon-Sanderson**: Quoted by Carr (6).

[6] **Carr, J. W.**: "The Starting Points of Tuberculosis in Children," a paper read before the Med. Soc. Lond., May 7, 1894.

[7] **Colman**: Brit. Med. Assoc., Newcastle, 1893.

[8] **Cornil and Babes**: *Les Bactéries*, Vol. II., p. 433.

[9] **Coutts**: *Brit. Med. Journ.*, 1901, August 10, p. 381.

[10] **Dobroklonski**: Referred to by Bovaird [4] (*a*) and by Northrupp (18).

[11] **Guthrie, L.**: "The Distribution and Origin of Tuberculosis in Children," *Lancet*, 1899, Vol. I., p. 286.

[12] **Heller**: *Deutsch. med. Woch.*, 1902, September 25, p. 696.

[13] **Holt**: "Tuberculosis in Infancy and Early Childhood, with special Reference to the Mode of Infection," *Med. News*, 1896, Vol. LXIX., p. 657.

[14] **Koch**: (*a*) Tuberculosis Congress, London, 1901. (*b*) International Conference on Tuberculosis in Berlin, *Brit. Med. Journ.*, 1902, Vol. II., p. 1885.

[15] **Kossel**: *Zeitschr. f. Hyg. u. Infektionskr.*, 1896, Vol. XXI., p. 59.

[16] **Lister**: *Brit. Med. Journ.*, 1901, Vol. II., p. 283.

[17] **Martin, S.**: Royal Comm. on Tuberculosis, 1895; referred to by Shennan.

[18] **Northrupp**: "Tuberculosis in Children, &c.," *New York Med. Journ.*, 1891, Vol. I.

[19] **Ponfick**: Referred to by Woodhead (22).

[20] **Shennan**: "Tuberculosis in Man and the Lower Animals," *Scottish Med. and Surg. Journ.*, 1901, September to October, p. 265.

[21] **Still**: *Brit. Med. Journ.*, 1899, August 19.

[22] **Woodhead, G. S.**: "Tabes Mesenterica and Pulmonary Tuberculosis," *Laboratory Reports, Roy. Coll. of Phys., Edinburgh*, 1889, Vol. I., p. 179.

[23] **Wyssokowiz**: Referred to by Northrupp (18).

VARIATIONS IN THE TECHNIQUE OF CATARACT-EXTRACTION AS PERFORMED IN SOME CONTINENTAL EYE-HOSPITALS.

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DURING 1902 I was fortunate enough to be able to visit a number of the principal eye-hospitals in Italy, Switzerland, Austria, Germany, Belgium, Holland, Poland, Russia, Denmark, Sweden, and France. I had the additional advantage of official introductions to the various Governments, which had been most kindly obtained for me by His Excellency the Secretary of State for India, at the courteous instance of my own Government. If there was one thing more than another which my opportunities impressed on me, it was the variety of method employed by different surgeons to obtain the same end. Nowhere was this extraordinary diversity of procedure more obvious than in the conduct of a cataract-extraction, and I have accordingly chosen that operation as the text of the following pages, confining my remarks to Continental experience,[1] and leaving home practice out of my paper for obvious reasons.

Each stage of the operation will be dealt with in turn.

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[1] The following are the names of the surgeons whose methods are alluded to in the present paper:—

Prof. Sbordone (Naples).
Prof. Businelli and Prof. Scellingo (Rome).
Prof. Otto Haab (Zurich).
Prof. Eversbusch, H.R.H. Duke Carl Theodor, and Dr. Zenker (Munich).
Profs. Fuchs and Schnabl (Vienna).
Prof. Pagenstioher (Wiesbaden).
Dr. Tacke and Prof. Coppez (Brussels).
Dr. Rogman (Ghent).
Prof. de Haas (Rotterdam).
Profs. Straub and Juda (Amsterdam).
Profs. Snellen, Senr. and Junr. (Utrecht).
Dr. Bouvin (The Hague).
Profs. Hirschberg, Greeff, and von Michel (Berlin).
Prof. Kanochi (Warsaw).
Prof. Krukoff, and Drs. Adelheim and Golowin (Moscow).
Prof. Schroeder (St. Petersburg).
Prof. Widmarck and Dr. Nordenson (Stockholm).
Prof. Csullstrand (Upsala).
Profs. Hansen-Grut and Bjerrum (Copenhagen).
Prof. Volckers (Kiel).

PREPARATORY MEASURES.

The Sterilisation of Instruments.—In the clinics which I have visited this is effected by (1) moist heat, (2) dry heat, or (3) the action of antiseptic solutions.

The favourite form of *moist heat* is boiling water to which some sodium carbonate has been added. A few use distilled water, either plain or after the addition of the sodium-salt. All the Continental surgeons I met admitted that the knife suffered from boiling. To minimise this Hirschberg, Nordenson, and others avoid placing it in the tray with the other instruments, and instead hold it for a short time in the boiling fluid. Others except the knife from boiling, and prepare it in other ways. Juda, for instance, soaks it for 20 minutes in a sterilised soap-solution, and then carefully washes it with sterilised wool and water. Very few seem to use steam to sterilise their instruments. I prefer its use to that of any other agent.

Dry heat has not a few advocates. Adelheim and Krukoff use it at 105° C., Nordenson at 150°, and de Wecker at 160°.

Very few trust to antiseptic solutions alone, but De Haas relies on absolute alcohol, and Schroeder on the biniodide. It is, however, a common thing to see a surgeon who uses one or other form of heat-sterilisation transfer his instruments into an antiseptic solution till such time as he is ready to use them. A variety of antiseptics are used for the purpose, amongst which I may mention several salts of mercury, for instance, the perchloride, the cyanide, and the biniodide.

A few operators have each instrument passed through boiling water (*e.g.*, Krukoff) or through a flame (*e.g.*, Lapersonne) before using it a second time, whilst Landolt takes care to have duplicates ready, so that it is never necessary to use any single instrument twice.

Personal Asepsis of the Surgeon.—Very many of the operators I met carefully washed their hands before each operation, and then soaked them in an antiseptic lotion. Some dried them on aseptic towels, others allowed them to dry in the air, while Landolt puts on loose, sterilised, linen gloves from the moment he finishes washing till he commences operating, when he throws them aside. His assistants do the same. Some operators go so far as to use specially-sterilised soap in tubes. Hirschberg puts on a sterilised over-all apron, and asks all who approach the operating table to do the same. Very few ophthalmologists cover the patient's face with sterilised cloths to protect their hands during operation. Greeff and Lapersonne both take this precaution. Landolt and Völckers confine female hair within rubber bathing-caps on the operating table. De Wecker ridicules all such measures as "l'antisepsie d'ostentation." As might be imagined, one saw many lapses from the grace of a perfect antisepsis in different clinics, but it would be ungracious to linger over them.

Choice of Assistants.—Some operators multiply the number of their assistants, giving to each one a small task, whilst others are careful to limit the numbers of hands which approach their patient. De Haas, Sbordone, and Rogman prefer to operate without any help from assistants, while the majority of surgeons accept the aid of one or, at the outside, of two helpers. As a general, but unfortunately not as an invariable, rule, the operator demands the same standard of asepsis from his subordinates as he practises himself. A few, like Hirschberg, will allow no one but themselves to touch their instruments, while some detail a special instrument-assistant to feed them as they require.

Some surgeons make a point of operating as soon as they reach their clinics with the double aim of avoiding carrying infection and of giving their best and freshest moments to the responsible work of the theatre. Others prefer to finish the wards and out-patients first, in order to know what is before them for the day.

Choice of Instruments.—Trousseau uses only one instrument for an extraction, viz., a knife. Masselon adds another, to wit, a pair of conjunctival forceps. Fuchs, whilst not otherwise limiting himself, omits the use of a speculum. Landolt, at the opposite pole of practice, not only keeps ready every instrument he may by any chance want, but also makes a point of having by him duplicates of any part of his armentarium which may be required more than once.

Preparation of Drops used.—Many surgeons dissolve their alkaloids in antiseptic solutions of different natures and strengths. De Haas uses $\frac{1}{1000}$ perchloride, Adelheim $\frac{1}{6000}$ cyanide, and so on. Trousseau and others prefer distilled water for a vehicle. Many sterilise their drops by heat. Hirschberg boils all his in special bottles made of a glass which, according to his claim, does not admit of chemical changes occurring between it and the alkaloids. He has a separate bottle of drops of each kind for each different patient.

Cocaine still appears to hold its own as the local anæsthetic for extractions, and it was quite the exception to find other alkaloids in use in any of the clinics I visited. Rogman favours tropacocaine, while Hirschberg recommends a mixture of cocaine and holococaine. These are the only exceptions to the use of cocaine I can recall.

PREPARATION OF THE PATIENT.

General Considerations.—Landolt keeps his patient in bed, or at least in hospital, for several days before an extraction, with a view to studying his state of health, preparing him for operation, and getting him used to his new surroundings. On the other hand, I have seen De Wecker take a cataract-patient on to the operation-table direct from the O. P. room. With a wide divergence in detail, most ophthalmologists conform, in their practice, rather to the more cautious habits of the first-named surgeon.

Cleansing of surrounding Skin.—De Wecker is averse to the use of soap, on the ground that it is irritating to the conjunctiva. Fuchs so far agrees with him as to avoid soap on the day of operation. Most surgeons, however, use it freely, either on the day of operation, on the preceding day, or on both. Immediately before an extraction most ophthalmologists wash the surrounding skin freely with one form or another of antiseptic lotion. Landolt prefers to use sterilised soap and water for the purpose, in order to avoid any irritation of the delicate skin of the parts.

Völckers keeps the eye covered for an hour before an extraction with a pad soaked in $\frac{1}{1000}$ solution of perchloride. Lapersonne speaks highly of Panas' method of closing the eye for 24 hours before operation with a dressing soaked in a $\frac{1}{250}$ solution of biniodide of mercury in sterilised oil. A few others leave an antiseptic pad placed over the eye from the time it has been got ready till they are about to operate, but otherwise I have met with no imitators of the more systematic efforts to sterilise the skin by dressings, which I have just described.

Treatment of the Lashes.—Most surgeons leave the cilia uncut, and thereby run no small risk of fouling their instruments thereon during operations, as I have had more than one opportunity of observing. A few operators, amongst whom one may mention Von Michell, of Berlin, cut the upper lashes only; others cut those of both lids, whilst I limit myself to dealing with those on the temporal half of the upper lid, thus protecting my knife from soiling at the expense of a minimum of mutilation. Schiotz and Nordenson epilate both lids entirely. Hansen Grut tried this plan, but gave it up on account of its painfulness.

Panas laid great stress on cleansing the lid-margins with pledgets of wool soaked in an antiseptic. Each lid is seized and held in turn for the purpose. Schroeder substitutes benzene for an antiseptic in carrying out this measure.

Cleansing of the Conjunctiva.—De Wecker is in a marked minority in considering that the conjunctival sac, provided its lining membrane is healthy, should not be interfered with before an extraction, for the great majority of ophthalmologists are of the opposite opinion. Some irrigate the cavity with an aseptic solution, which acts purely mechanically as a sweeper, or which at the most exerts a solvent action on flakes of mucus. Thus we find that Kanochi, Krukoff, and Völckers employ normal saline solution for the purpose, whilst Landolt uses a sterilised solution of boric acid, and Schnabl a one-per-cent. lotion of bicarbonate of soda. Others, however, prefer antiseptic irrigations of various natures and strengths. Von Michell uses perchloride lotion at $\frac{1}{5000}$; Fuchs employs the same solution at $\frac{1}{4000}$; Sbordone, Karl Theodore, Haab, Pagenstichter, Hirschberg, and Greeff select a strength of $\frac{1}{5000}$, whilst Hansen Grut and Bjerrum prefer $\frac{1}{6000}$. Adelheim, Trousseau, and Rogman irrigate the sac with various strengths of the bicyanide solution. Lapersonne favours the biniodide. Nordensen and others the oxycyanide, and so on. My own preference is for a $\frac{1}{5000}$ solution of freshly-prepared Chinosol, but at the same time I rely rather on the mechanical than on the antiseptic action of the fluid. In this respect I believe I am at one with the great body of European ophthalmologists.

Treatment of the Lachrymal Passages.—There is considerably more difference of opinion here than we met with under the last heading. If suppuration be present, all are agreed as to the necessity for preliminary measures, but it is otherwise when we come to consider the bearing of old chronic obstructions, where the sac contains a clear fluid, or where obvious signs of disease are conspicuous by their absence. De Wecker implies his scepticism as to the need of excluding such cases from the theatre, and many reliable surgeons, amongst whom I may mention Hansen Grut and Bjerrum, consider that, unless there is definite evidence of disease, it is better to let the tear-conduits alone. Völckers, on the contrary, considers that "diagnostic irrigations" of these passages should be a routine part of the examination of a patient before he is submitted to an extraction. Kanochi and Nordenson go even further, and always syringe out the lachrymal passages before a major eye-operation, in order to remove any septic matter they may contain. A few surgeons are careful, with the same end in view, to empty the sac by firm digital pressure.

Völckers and many others, if in doubt as to the state of the passages, extirpate the sac before performing an extraction. Fuchs either extirpates, or else splits the anterior wall, and plugs the cavity. Haab prefers temporarily to obliterate the puncta, whilst my own preference, in mild cases, would be to close the canaliculi for the time being by means of small sutures.

THE OPERATION.

Position of Operator, &c.—Most Continental operators whom I saw at work stand behind for the right eye and to the left front for the left eye. I noticed one surgeon who stood at the right front for left eyes. Several ambidextrous German ophthalmologists work on both eyes from in front, thus reversing the procedure followed by English operators in India, who usually stand behind for both eyes.

In the majority of clinics extraction is performed on a table, but some surgeons prefer a low couch, and others a sloping chair. A few perform secondary discissions with the patient sitting opposite to them, upright, in a straight-backed chair.

Most surgeons prefer to operate by daylight, but there are some, both in Great Britain and the Continent, who elect, as do De Wecker and Masselon, to operate by artificial light. Snellen's blackened operating room, with its wide window-space covered by black blinds, any one of which can be drawn up separately, is probably known, by repute at least, to all. It is the rule to see the patient laid parallel to the window, with the eye under operation on the same side as the source of light, but there are those who prefer to place the patient with his feet towards the source of light, whilst they stand behind his head.

Those who use artificial light commonly employ a bull's-eye electric lamp, which is entrusted to a special assistant. In the new clinic at Hamburg, I saw a very beautiful little electric forehead-lamp worn by the surgeon. Its many advantages are too obvious to need mention.

Management of the Patient.—Some ophthalmologists take a great deal of trouble with their patients in the few days before an extraction; they train them to open their eyes widely, to close them gently, to look down, and so on. Hansen Grut is especially careful about this training. During the operation he asks the patient to hold up his hand and keep watching it. Many surgeons adopt the same manœuvre; others again simply bid the patient look towards his feet, whilst an assistant aids the effort by gently pinching his toe; yet others ask a bystander to raise his hand to assist the patient in fixation, or to hold up a light or other bright object for the same purpose. My own plan is to operate with the patient's feet towards a window, and to bid him look steadily at the light. Adelheim and de Haas make a great point of putting cocaine in both eyes, instead of in the operated eye only. I have seen this done in London also, and all who do it say that it helps to keep the patient quiet. Krukoff and Kanochi, with a similar object in view, close the opposite eye during an extraction.

If a patient becomes restless and frightened, some operators try to reassure him by talking quietly to him; failing this, many slap him sharply, and the effect is often magical in bringing a nervous subject back to self-respect. Tacke gives a timid patient chloroform. My own practice, which I cannot too strongly recommend, is, if I find a patient getting out of hand, to delay the operation for 15 minutes, and give a hypodermic of morphine. The result is magical, but it is *essential that the preparation used should be fresh and active, and that the injection should be rubbed in,* so as to secure an early and powerful action. Of course the state of the kidneys must have been ascertained beforehand.

The Incision.—The great majority of European operators make an upward incision, which they keep in the limbus throughout its whole extent. A few, like Fuchs and Pagenstocher, prefer to add a small conjunctival flap. Professor Snellen (junior), of Utrecht, has adopted a sideral incision wholly outside the limbus, and he denies that this procedure increases the risks of vitreous or iris-prolapse. Adelheim, of Moscow, is the only other surgeon I have met who adopts this method of opening the eye. Völckers, of Kiel, who makes a limbus-incision for simple extractions, modifies his method for the combined procedure, and while making his puncture and counter-puncture in the sclerotic brings out his blade at the summit of the clear cornea, thus irresistibly reminding one of Von Graefe's "modified linear extraction." Italy was the only country in which I saw "purely corneal incisions" in use. Professor Sbordone, of Naples, and Professor Scellingo, of Rome, were, indeed, the only two operators in Europe whom I noticed adopting this rare method of commencing an extraction. I could not help observing that, in Sbordone's cases at least, the low placing of the incision led to increased difficulty in delivery of the lens.

Another curious feature of Scellingo's technique is that he invariably makes a downward section, believing that it is much easier to deliver through a downward than through an upward cut. Schroeder, of St. Petersburg, and Nordenson, of Stockholm, prefer a downward section in the deeply-sunken eyes of Mongolian patients, in whom they find it difficult to make an upward section. In all ordinary cases, however, they follow the usual rule of working in the superior quadrant of the eye.

Rogman, of Ghent, has two peculiarities in the first stage of his operation, viz.:—(1) That he seizes the conjunctiva, with his fixation-forceps, just below his expected point of counter-puncture, instead of in the usual position, and (2) that he appears to cut out with a straight pull instead of sawing out as others do. He is one of the most skilful operators I have ever watched at work.

Method of lacerating the Capsule.—For this purpose the cystitome remains the favourite instrument. Indeed, I saw it in use in nearly every country of Europe. The use of capsule-forceps, though warmly championed by Fuchs and by De Wecker, does not seem to have recommended itself to the profession. Sbordone, of Naples, and the Duke Carl Theodore and his assistant, Dr. Zenker, were the only other ophthalmologists, beside the two just mentioned, whom I saw using this instrument. De Wecker admits that it is a difficult instrument to work with and that it easily gets out of order. Trousseau, of Paris, lacerates the capsule with his knife-point during incision. This procedure has advocates in England, and I have used it myself in dealing with tough Morgagnian capsules, but it is not likely to be generally adopted, since it tends to sacrifice precision without offering any substantial gain in return. Nowhere out of India have I seen the capsule lacerated with a Bowman's needle before the incision is undertaken, and yet for precision and safety in effecting the needling, for the easy sterilisation of the instrument used, and for the valuable information thus acquired as to the consistency of the cataract, and the size of its nucleus (enabling us to graduate our size of incision accordingly), this method has to my mind no equal.

Before leaving the subject, one must mention Landolt's method, which, if rather quaint, has at least the virtue of thoroughness, a virtue which pervades all this surgeon's work. He uses two sickle-shaped cystitomes, one of whose curves looks to the right, whilst the other looks to the left. These instruments are introduced in turn, and swept across the capsule, which is thus freely lacerated in opposite directions by their curved cutting blades.

THE QUESTION OF IRIDECTOMY IN CATARACT-EXTRACTION.

This question has been so long discussed that some apology may be needed for bringing it up again. I trust that the wide divergence of opinion which still exists on the subject may be taken as some justification of my action in doing so, especially as my object is rather to compare and contrast the opinions of others than to press forward any views of my own.

The cataract-operators whom I have met may be divided, for convenience' sake, into three classes, viz.:—(1) Those who always select the combined operation, if possible; (2) those who prefer to operate by the simple method, if they can; and (3) those who, possibly with a leaning for one or the other of the above methods, make a point of selecting the procedure to be adopted in each case, according to certain rules of their own.

It might be thought that in this, as in other cognate matters, nationality would exercise a considerable influence over surgical opinion, and indeed so far as Italy and Russia are concerned this seems to be actually the case. In Rome and Naples at least the bias is strongly turned towards the simple operation, whilst in Warsaw, Moscow, and Petersburg, I only met one single surgeon who shared the Italian views; all the rest looked on the performance of an iridectomy as almost a routine part of an extraction-operation which should never be omitted if it could be helped. In no other country of Europe, so far as I am aware, can a similar uniformity of practice in this respect be found. In Belgium one finds Tacke and Coppez, of Brussels, both strongly in favour of the combined operation, whilst not 40 miles away, at Ghent, is Rogman, who rarely departs from the simple method of extraction.

In Holland the undoubted bias of opinion is towards the combined procedure. De Haas, of Rotterdam, and Bouvin, of The Hague, never omit an iridectomy; Straub, after carrying out careful comparative series of operations, has come to nearly the same line of practice, as has also Juda, of Amsterdam. And yet, within a few miles of any of them at Utrecht, the Snellens only resort to the removal of a portion of iris under exceptional circumstances. In Germany, again, one meets with all shades of opinion, and the same may be said of Sweden, but in the latter country the leaning towards the combined operation is more marked, and the simple method has no ardent exponent that I know of. In Paris one finds Landolt and Lapersonne strongly impressed with the need of considering iridectomy a routine step in cataract-extraction, whilst working side by side with them in the same city are two of the most ardent advocates of the simple method that I met with anywhere in Europe. I, of course, refer to De Wecker and Trousseau. Strangest of all was it to find in the one, and I believe the only, recognised eye-hospital of Denmark two surgeons working under the same roof, one of whom always performs the combined operation, whilst the other equally invariably aims at completing his extraction without an iridectomy; and yet this is true of Hansen Grut and Bjerrum.

The list of surgeons who, even in my own small personal experience, look on iridectomy as an essential and not-to-be-omitted step in cataract-extraction, makes impressive reading. It includes Schroeder, Adelheim, and Kanochi, each from a great centre of Russian thought; Landolt and Lapersonne, of Paris, Schnabl, of Vienna, De Haas, of Rotterdam, with a record of 2,100 extractions in 40 years of work, H.R.H. Carl Theodore, of Munich, whose total of extractions has passed 3,000, and others besides. Nor is this all the support accorded to "invariable iridectomy." Very significant was Fuchs' statement to me that he "always employs the combined operation for his private patients," for though he has strong leanings towards this method in his hospital practice he does not invariably use it there. I may add that his co-professor, Schnabl, has only settled down to routine iridectomy after fully trying the simple operation. Pagenstícher, whilst himself selecting the procedure which seems best for each individual case, strongly advises all beginners invariably to include iridectomy in their operation for extraction. Lastly, there are not a few surgeons, both on the Continent and at home, who in cases in which there is only one eye, or in which other indications for special care exist, not merely include an iridectomy in their operative measures, but perform that step as a preliminary sitting. Strangest of all is it that this precautionary measure should be adopted by two of the most ardent advocates of the simple operation to be found in Europe, and yet this is true of De Wecker and Snellen (junior). Lapersonne, who, as has already been said, looks on the removal of a portion of iris as a routine step in his extractions, does iridectomy as a preliminary operation in all cases of immature or complicated cataract, and in those whose general health is bad.

It will be of interest to turn now to the opinions of some of the advocates of the simple operation. Hirschberg prefers to operate by this method, if possible, but prides himself on individualising most carefully in each case. Trousseau's only indication for iridectomy, in extraction, is the presence of a cough. De Wecker names two indications, viz.:—(1) Difficulty in immobilising his patient, and (2) immaturity of the cataract. Rogman likewise names two, which are (1) a tendency to iris-prolapse after completion of the operation, and (2) an injury to the iris during the performance of the section. Bjerrum shares Rogman's first indication, which indeed is his only guide for iridectomy, provided he can keep his patient quiet after operation. Snellen (junior), who uses pilocarpine drops after operation, never performs the combined operation at one sitting if he can avoid so doing. For the extraction of immature, hypermature, and complicated cataracts he considers that an iridectomy is called for, but he performs it at a preliminary séance. In no case would he venture to operate without iridectomy on a patient whom he could not immobilise after operation. One Russian surgeon and the Italians conclude my list of Continental advocates of the simple method. I may mention that the use of myotics, either after or before extraction, is by no means universal in the practice of those who omit an iridectomy.

Apart from the usual arguments, viz., æsthetic considerations, the retention of a measure of accommodation, and the perfection of the light-regulating diaphragm advanced in favour of simple extraction, there are four contentions which I have often heard put forward by the Continental advocates of the method. These are (1) that prolapse is a comparatively uncommon complication; (2) that it also occurs after extraction by the combined operation; (3) that it is not really a very serious matter; and (4) that the operative measures for its relief are not very dangerous in their nature. Leaving the more time-honoured considerations aside, as they have been so fully discussed elsewhere, I wish to say a few words on the above four headings.

(1) A table showing the admitted percentages of prolapse of the iris occurring in the practice of a number of different advocates of the simple operation could not fail to be of great

interest, and an effort to compare and harmonise the results obtained would be likely to lead to valuable conclusions. I endeavoured to collect the requisite information at the various clinics I visited, but was obliged to give up all hope of doing any good, for I found that, while the figures quoted to me differed widely, there were but very few instances in which they had been derived from a painstaking survey of accurately kept statistics. There is one point, however, on which an overwhelming majority of Continental ophthalmologists appear to have clearly come to a decision, viz., that it is not possible to exclude prolapse of the iris even after the most careful selection of cases or by means of any known technique.

(2) Probably everyone will admit that in a certain number of cases the cut edges of the iris become involved in the section during healing, and even that, in a further small percentage, an actual prolapse of the same portions takes place. The frequency of these accidents can be greatly reduced by paying strict attention to the thorough replacement of the iris after delivery of the lens. It has been my experience that, even when prolapse does occur under these circumstances, the accident is of a much less serious nature than when occurring after simple extraction. In this opinion I have been fortified by the experience of a large number of Continental surgeons, amongst whom were some of the advocates of *extraction sans iridectomie.*

(3) and (4) We may take the dangers of prolapse of the iris and the gravity of the measures for its relief together. I look upon prolapse of the iris as a most serious complication of the after-course of an extraction, and have been astounded to hear surgeons of experience express a different opinion. Again in all marked cases of prolapse I have been led to look on the opening of the section and the free removal of the caught iris as absolutely necessary for the safety of the eye, but, at the same time, as measures involving no small hazards. Even when the prolapse is of small extent and gives rise to no urgent symptoms I am far from regarding its occurrence without misgivings as to the future. It then becomes a matter of balancing the risks of action and inaction; even when action is decided upon it may be limited to a cauterisation of any protruding portion of the iris. Here again have my cherished

beliefs been rudely assailed, and I have been told that it is very seldom necessary to interfere with a prolapse, and that even when such interference is necessary it may be undertaken with a light heart. Such are not, however, the views commonly held on the Continent; and from a large number of ophthalmologists, from east to west and from north to south of Europe, I received it, in no uncertain terms, that prolapse is to their minds a very dangerous complication, and that the operation of opening the section, removing the portion of impacted iris and freeing the membrane is, in their experience, often necessary, and is farther a very serious addition to the risks of the first operation. And yet, with all its added dangers, it is not difficult to see that surgeons everywhere still hanker after simple extraction. I have already given the indications accepted by some as a calling for a departure from their usual practice of never resorting to iridectomy, if they can help, and now turn to review the conditions which sometimes induce advocates of the combined operation to depart from their beaten track. Eversbusch, of Munich, performs a simple extraction only if the patient is in good health in all respects, and if the chamber is deep, the iris active, the tension normal, and the tunics and vitreous healthy. Völckers occasionally ventures on the same method when all the above conditions are fulfilled, but even then he finds himself unable positively to exclude prolapse, and hence has a strong leaning towards invariable iridectomy. Juda, while following generally the same lines as Eversbusch, adds an extra condition, viz., the youth of the subject. Hirschberg and Pagenstiche insist most strongly on the necessity of minutely and carefully examining each case, in order that each may be decided on its own merits. Speaking generally, their indications for selecting the simple method seemed to agree with those of Eversbusch, and in the same category I may also include Greeff and Coppez. I omitted to mention that Eversbusch considers cases of soft cataract as particularly suitable for the exhibition of the operation without iridectomy.

Widmarck performs the simple operation in mature, and the combined in immature, cases. He keeps the former five days in bed after operation and the latter only two days. He has twice met with sudden death from syncope in old patients on their first rising from the horizontal position in which they had been kept some days after an extraction. He, therefore, now always prefers the combined operation for old people. Nordenson, with characteristic caution, only resorts to a simple extraction when, in a binocular case of cataract, he has already obtained a good result by means of the combined operation on the other eye, and only then if all the indications are favourable. Fuchs considers that the results of a simple extraction are the most perfect obtainable, but that at the same time the risks run are distinctly greater than when a portion of iris is excised. Where all the indications are distinctly favourable, he would omit an iridectomy, with the proviso that this plan should be altered if the course of the operation should reveal an undue rigidity of the iris, a tendency of that membrane to prolapse after the operation is completed, or any other unforeseen abnormality. He especially prefers the omission of an iridectomy in the young on account of the better cosmetic results.

Time will not permit me to dilate on a number of other points, which, though of minor importance, are nevertheless of considerable interest, inasmuch as they illustrate the extraordinary number of ways that different surgeons take to do the same thing. Some operators perform iridectomy with straight scissors, others with curved, others again with cross-action scissors, and so on. Some make a point of always removing a large piece of iris; not a few hold that the smaller the iridectomy the better, and many do not seem to think that the matter is of any importance, if the looker-on may judge by their practice. Yet another detail. Most operators cut the iris off with one bold closure of the scissors, whilst a few are careful to carry their section right up to the iris-attachment by means of two or more snips. The greater number of surgeons will probably agree with me that if an iridectomy is to be done at all, it is both easier and safer to do it before the escape of the lens. Bjerrum, however, holds the opposite view.

REMOVAL OF THE LENS.

The methods in vogue for this purpose are very numerous, and will be better understood if we divide the subject up into two heads, viz. :—(1) the delivery of the nucleus, and (2) the removal of the cortex.

(1) The nucleus may be delivered (*a*) by instruments alone, (*b*) by digital manipulation alone, or (*c*) by a combination of the two.

(*a*) Sbordone delivers by the aid of two knife-handles; Schroeder, De Haas, and Rogman by two spoons; Lapersonne with a curette and spatula; others use their fixation-forceps and a curette attached to the opposite end of the knife-handle; this last method saves multiplying instruments, but is an objectionable practice. The retention of the speculum is almost a necessary condition for purely instrumental delivery. A few operators follow Hansen Grut in using only one instrument, which is applied below, the lids being separated and the patient told to look down steadily.

(*b*) Fuchs delivers by digital pressure, exerted through the lower lid on the inferior portion of the cornea; the upper lid is held out of the way. Landolt and Masselon use bi-manual digital pressure, by working with a thumb or finger through each lid; they thus obtain pressure and counter-pressure. Landolt, of course, removes the speculum before employing this manœuvre, while the other two do not use one at all.

(*c*) Yet other surgeons combine the two previous methods; they remove the speculum, if they have employed one, and getting an assistant to depress the lower lid, they use an instrument below, while a finger above supplies the counter-pressure. De Wecker is one of those who use this manœuvre.

(2) Not a few methods are adopted to rid the chamber of cortex. Many employ massage applied through the lower lid, the upper one being raised out of the way. Among such are Kanochi, Krukoff, Sbordone, Hansen Grut, Fuchs, and Lapersonne. Others prefer bi-manual digital massage for the purpose, a finger or thumb working through each lid. Trousseau, Landolt, and Masselon, who, as we have already seen, employ this latter method to express the nucleus, continue thereafter to use it to rid the chamber of débris. De Wecker also uses it in this stage of the operation.

Of those who deliver the nucleus by the aid of instruments, a large percentage continue to use those same instruments for the evacuation of any cortex which has been left behind; this manœuvre, which we may speak of as "instrumental massage," consists in stroking the cornea in the direction of the incision with a curette, scoop, spoon, or other instrument. I saw Schroeder, Rogman, and De Haas using this means, which Csullstrand and Von Michell also employ. A few surgeons introduce a curette, scoop, or spoon into the chamber to remove therefrom cortical masses which less direct methods have failed to displace. Fuchs, Kanochi, Schroeder, De Wecker, and others resort to this method, though most of them will probably agree with the last-named in considering it a dangerous procedure. Most, but not all, of the operators, who enter the anterior chamber in this hazardous way, are very careful to sterilise afresh the instrument introduced before each new entry of the chamber.

De Wecker holds strongly that all lens-débris should be evacuated, on the ground that masses left behind swell up, and favour prolapse. For the same reason Lapersonne is most patient and painstaking in his efforts to clear the anterior chamber of all lens matter, and to this end he allows the aqueous to reform repeatedly, and performs massage again and again. Gullstrand, Rogman, De Haas, and others avoid pushing the cleansing of the chamber too far, fearing the grave dangers which over-interference is liable to entail; not least among such is loss of vitreous.

I have kept till last a mention of M'Keown's method of intra-ocular irrigation, which appears to me to have never been sufficiently tried by ophthalmologists. It has given me far the best results of any method I have tried, and I have had ample opportunities of using all those commonly advocated. It is safe, clean, and effective, provided that the most ordinary care is taken, and after using the method on over 800 cataracts in eight months, it appears to me to be the one important step in advance made recently in the treatment of cataract. I am glad to be able to say that Fuchs, Haab, Nordenson, and Hansen Grut are now giving irrigation a fresh trial with instruments I had the pleasure of obtaining from Belfast for them. This paper is no place for farther details of the subject, which I have already dealt with fully elsewhere. (*Archives d'Ophthalmologie*, 1903, and *Ind. Med. Gazette, April* 1903.)

The Speculum.—The models of this instrument which one saw in use are too numerous to be even mentioned. Some insert it with its arms over the nose, others keep them on the temporal side. Some entrust the blepharostat to an assistant, and lay great stress on the *rôle* the latter plays in avoiding undue pressure on the eye, whilst others leave the instrument quite alone and untouched. Some allow the speculum to remain in throughout the operation; others remove it as soon as the incision is made; yet others take it out after the iridectomy, or after the laceration of the capsule; lastly, as mentioned elsewhere, there are those who never use a speculum at all.

METHODS OF BANDAGING.

Hjort leaves both eyes unbandaged and uncovered. Masselon closes the operated eye alone, using for the purpose a narrow vertical strip of gauze soaked in collodion. De Wecker applies the lightest possible dressing to the operated globe, and keeps the pad in place by the aid of two narrow strips of collodioned gauze. Krukoff, Straub, Bouvin, and Hansen Grut all limit themselves to closing one eye only. The majority of operators still, however, close both. The period of closure varies within too wide limits to admit of an average giving any useful information. The operated eye is opened in from 24 hours to a week or 10 days, the opposite one being released at any time from 24 hours onward. A few operators open both together, while more keep the wounded one closed for a few days later than its fellow. Some keep the bandages on at night after they have been discontinued by day.

The forms of bandage which one sees employed for extraction cases are legion. They include rollers, figures-of-8, many-tails of numerous patterns, triangular bandages, modified night-caps, starched bandages, &c., &c. Then there are shields of many kinds which some use as substitutes for, or in addition to, bandages. These are single or double, are fastened on with tapes, with plaster, or with bandages; they are made of gauze-wire, of vulcanite, of celluloid, or of some form of metal; and, lastly, they differ widely in shape; some, like Wolff's pattern, have sloping upturned edges to save the patient's face, whilst others, like Snellen's metal model, are purposely provided with sharp margins, in order that these may cut into the subject if he attempts to interfere with his bandage; they are in fact intended to serve as timely reminders, lest he forget.

Some surgeons aim at providing firm pressure by means of their bandages, others only draw theirs sufficiently tight to keep the dressings in place, and so on. Some combine shields and bandages, or substitute the former for the latter, as soon as possible. A few, if in doubt as to the state of the lachrymal passages or conjunctiva, use a shield throughout to avoid retention of secretions, whilst others dispense with all covering of the eye under such circumstances.

In the matter of dressings, too, there is great variety, both as to their quantity and their nature. Between De Wecker's thin layers of gauze and the elaborate dressings of some other surgeons there are many intermediate stages, but in one respect most are agreed, for all who can do so sterilise them in an autoclave. A layer of lint or gauze is usually applied directly over the lids, and is succeeded by a pad of sterilised wool. Many surgeons soak their dressings in one form or another of antiseptic. Straub is peculiar in using a dressing of sterilised modified zinc-ointment, which, he claims, restrains secretion and keeps the eye dry. Landolt and Hirschberg not only keep each patient's dressings quite separate in the steriliser from those of any other, but are also careful to keep each constituent part of each dressing by itself.

Lastly, in closing, I may mention a variation from the standard type, which surprised me more perhaps than anything else I saw abroad. I refer to the practice of two well-known surgeons, Schnabl and Sbordone, of operating on both eyes for cataract at one sitting when possible. It is a practice which the Government of India forbids its officers to indulge in in the course of their Government work.

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PERIPHERAL NEURITIS OF THE ALCOHOLIC TYPE.

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THE problem of the exact causation of the so-called alcoholic neuritis remains practically where Dr. E. S. Reynolds left it in his admirable paper read at the meeting of the British Medical Association at Cheltenham in 1901. That the type of multiple neuritis with which all are now familiar may be due to arsenic alone is generally conceded. The experiences which many have had with the drug in the treatment of chorea is sufficient to remove any doubt upon this point; though it is certainly curious, as Sir William Gowers[1] has pointed out, that the trouble does not more often follow the prolonged administration of Fowler's solution in combination with bromides in epilepsy. That the disease may be due to arsenic in very small doses ($\frac{1}{8}$ grain per gallon) when taken with beer has been fully established by the Manchester epidemic, and there seems every reason to believe that it was the conjunction of the two poisons rather than either singly which gave rise to the outbreak. That it may be due to alcohol alone, however, is considered by many to be by no means so certain; indeed, alcohol would now seem to be regarded as of quite secondary importance in the ætiology of the disease.

This very marked change in the conception of the chief causative factor is the direct outcome of the Manchester epidemic. Before that, the drinking of anything other than ardent spirits was not deemed sufficient to produce an attack, and none of the authorities, from Lettsom downwards, appear to have recognised any other cause. I well remember how, in the pre-arsenical days, the women coming with peripheral neuritis to the Out-Patient Room at Queen's Square would stoutly maintain that a few daily glasses of beer constituted their only alcoholic indulgence, and how the attending physician, in his most soothing and sympathetic tones, would say:

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[1] *Royal Medico-Chirurg. Soc. Transactions*, LXXXIV.

"But when you feel weak, perhaps you take a little drop of spirits ?" When the coy reply came in the affirmative, how satisfied we all felt that the real cause had been elicited! But the present-day tendency to embrace and hold fast to arsenic, while altogether discarding alcohol, though natural perhaps at the time of the Manchester epidemic, ought not to obtain in the clearer atmosphere into which we have now passed; for it is in reality merely the substitution of one partial truth for another, neither of which can be made to explain all the facts.

Indeed, no explanation of these facts can be regarded as in any degree satisfactory which fails to include, not only both alcohol and arsenic, but also some other factor or factors, of the exact nature of which only this can be said, that they bear no ascertainable relationship either to the one or the other. There are several, if not many, varieties of multiple peripheral neuritis of the alcoholic type that are due to causes which have so far altogether defied recognition.

But, first, let us consider the question of the responsibility of alcohol alone. Against the power of uncontaminated alcohol to give rise to the symptoms, it is urged that intemperance is very common, while multiple neuritis is very rare; and that, further, in a great many places where the consumption of whiskey is conspicuously great, the disease is conspicuously infrequent. In Glasgow, for example, where probably more whiskey is drunk per head of the hospital population than anywhere else, so keen, careful, and skilful an observer as Sir William Gairdner,[1] with his unrivalled opportunities for observing, was unable to vouch for more than ten cases at the outside in seventeen years. It is stated, moreover, that the experimental administration of ethyl alcohol to animals has so far failed to give rise to any symptoms resembling peripheral neuritis.

In favour of the alcoholic factor there is the important consideration that up to the time of Dr. Reynolds' brilliant work all those who had recorded such cases were agreed in attributing the disease to excessive consumption of spirituous liquors; and considering that a list of the observers contains such names as Wilks, Westphal, Broadbent, Dreschfeld, Charcot, Gowers, Buzzard, Thomsen, and Déjérine (to mention but a

[1] *British Medical Journal*, October 12, 1901.

few) it must be allowed that the evidence in favour of alcohol is very strong.

It is, however, impossible to disregard the weight of the argument which reminds us of the enormous prevalence of alcoholic excess, and of the comparative rarity of alcoholic neuritis; and it must be admitted that, though ethyl alcohol may provide a powerful, and possibly an essential, predisposing or contributing cause, it is, nevertheless, of itself insufficient to determine the disease. Hale White,[1] in a paper dealing with "Cirrhosis of the Liver," the other classical example of the effects of intemperance in spirituous liquors, draws a useful distinction between alcohol and alcoholic drinks, and suggests that there is in the latter some morbific, but elusive, element which is not present in the former.

In the case of multiple neuritis we are asked to believe that this element is arsenic, and that traces so minute as to evade ordinary tests may, nevertheless, suffice to produce the disease. It is the fashion to hold arsenic responsible for many things (cancer is its latest burden), but I have not yet heard it suggested that it has any hand in the causation of hepatic cirrhosis, so that if it be true that these minute traces which are assumed to be present in whiskey are really answerable for alcoholic neuritis, we must look for some other elusive element in the liquor to explain the occurrence of alcoholic cirrhosis.[2] But when we have both a poison and a soil to consider, it is surely unreasonable to expect any satisfactory solution to a problem from a consideration of one of them to the exclusion of the other. There are a great number of people, especially men, who drink the equivalent of a bottle of whiskey a day and who yet fall victims neither to neuritis nor cirrhosis, and their escape is certainly not due either to any especial moral merit on their part, or to any special demerit on the part of the whiskey. The factor which determines their immunity is purely physical; and although I am not at present prepared to affirm that this physical superiority is the result of climatic influence, it is at least highly suggestive, that the two diseases in question are so uncommon as to be rarities in those very parts of the United Kingdom where whiskey is most abundantly consumed. Given, then, a suitable human soil, there seems no more reason to doubt the power of alcohol in the form of ardent spirits, uncontaminated with arsenic or other mineral poison, to produce alcoholic neuritis, than there is to doubt the power of the same poison acting under similar circumstances to produce alcoholic cirrhosis.

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[1] *British Medical Journal*, March 7, 1903.

[2] The late Dr. W. B. Hadden relates in *Trans. Pathol. Society*, 1885, two cases of "alcoholic paralysis" which came to autopsy, and another in 1887. In all three the liver was diseased. In one it was "in an advanced cirrhotic condition"; in another it was "in a condition of advanced cirrhosis and fatty degeneration," and in the third it was large, pale, heavy, and finely granular."

But even with the question of alcohol *versus* arsenic settled, there remains another problem in connection with the causation of this type of the disease which is deserving of notice. There are a certain number of cases conforming absolutely to the type recognised as alcoholic for which, even after the most careful enquiry, no recognised cause can be ascertained.

In the paper which he read before the Royal Medico-Chirurgical Society, Dr. Reynolds[1] said that "the only forms of peripheral neuritis associated with great pain on muscular pressure are the alcoholic, the arsenical, and that form found in beri-beri." This statement is, I am convinced, a very much less accurate one than most of those which emanate from Dr. Reynolds; and Dr. Frederick Taylor, in discussing the paper showed very clearly that it was not in accord with his experience, by relating four instances of peripheral neuritis associated with tenderness of the calves, none of which were due to any of the above-mentioned causes. And not only are there cases of a similar nature recorded by others, but there are instances which conform to the alcoholic type, not only in the tenderness of the calves, but in practically every detail of the clinical picture, where nevertheless the recognised causes are surely absent. Such a case is recorded by Dr. Parkes Weber,[2] of a woman after abortion, in whom arsenic, alcohol, beri-beri, and sepsis could all be excluded; and the author quotes Bernstein,[3] as stating that this type of the disease may be due to so prosaic a matter as chronic constipation. It has, in fact, been the fashion in the past to look for an explanation of the disease too exclusively to alcohol, and it is now the fashion to rely too exclusively on arsenic. There are unquestionably cases which cannot be brought under either category. They are not many, perhaps, and there seems at present no explanation of those that do occur; but a general recognition of the fact of their occurrence is the necessary prelude to the study and ultimate solution of the problem which they present.

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[1] *Roy. Med.-Chirurg. Transactions*, Vol LXXXIV.

[2] *Barts.' Hospital Reports*, Vol. XXXIV.

[3] *Wratsch*, 1898, No. 29.

I plead therefore for the re-admission of alcohol on terms of equality with arsenic into the field of ætiological probabilities, with a strong recommendation in favour of some other cause or causes to the identification of which there is at present no evidence to point. In support of the former, for which Dr. R. T. Williamson[1] has already raised his voice, I desire to record a case in which arsenic could be positively excluded, one, moreover, which is possessed of some features of considerable clinical interest; and, in defence of the latter, to refer briefly to a case for which none of the causes at present considered adequate could after careful investigation be assigned.

The first case, which I saw in consultation with Dr. Septimus Sunderland, was that of a lady aged 40 who had been drinking to excess for some ten years. Eighteen months before I saw her she had had an attack of peripheral neuritis, ushered in by maniacal symptoms, which were followed in three weeks by pains in the legs and extensor paralysis in both feet. At the end of three months she recovered sufficiently to get about again, and immediately recommenced her drinking habits. Owing to the terrible scenes which ensued upon her husband's efforts to restrain her, he was compelled to relinquish the attempt, and when I saw her she had been taking about six bottles of whiskey a week, with an occasional bottle of stout, for over a year. All of these had been openly provided for her consumption, and owing to the fact that she seldom went out at all, and never unaccompanied, it is certain that she was not supplied from any other source. The whiskey and the stout in question have both been examined for arsenic, and in both cases the result has been negative.

At the commencement of the present attack she was in a state of stupor, with double internal squint, and passing both urine and fæces under her. The control of the bladder was regained after a few days, concomitantly with an improved mental condition, but the fæces remained uncontrolled up to the time when I saw her, fourteen days later. Six days before my visit she began to lose power in the muscles of her hands and back, so that she tended to drop things and was unable to sit up. Some tenderness developed in the muscles of both thighs, and the legs became numb and insensitive. At the date of my visit her mental condition was said to be distinctly improved, but her ideas as to time and place were very defective; she had no memory whatever for recent events; she imagined there was a baby in bed with her, and she was by no means sure of the identity of her husband or her nurse.

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[1] *Lancet*, January 26, 1901.

The appearance of the eyes was such as to suggest a "cast," but the muscles acted without weakness in all directions and the fundi were normal. There was, however, very decided nystagmus, not only on lateral deviation, but also on looking both upwards and downwards. The arms presented the characteristic wrist-drop appearance, with the hand in the "accoucheur's" position, but the muscles were not wasted. The paralysis of the affected extensors was complete, but the upinator longus was not involved. The arm-jerks were, however, absent. There was no pain or tenderness in the arms, and no other affection of sensation.

The muscles on the front and outside of each femur were very flabby and very weak, so that when the limbs were placed with the knees raised she was unable to prevent them from falling inwards towards one another. Pressure on these muscles caused pain. The hamstrings were unaffected, both as to power and sensation. The knee-jerks were absent; but so far from there being any weakness in the muscles below the knee, this portion of the limb on both sides was firm and strong. The foot could be dorsi-flexed against considerable resistance, and all the other movements at the ankle-joint were well performed. The plantar response was present and was definitely flexor. From the feet upwards there was, however, very considerable impairment of sensation to pain and touch, but not to temperature. The psychical state made it impossible to ascertain the degree and distribution of this impairment with any approach to accuracy, but it was fairly complete and reached as far as the knee and probably a little higher. There was no abnormality of skin, hair, or nails; no suffusion of the eyes, no laryngitis, no diarrhœa; nothing, in short, to suggest an arsenical element in the causation. The viscera, so far as could be ascertained, were normal. Insomnia was a pronounced and troublesome symptom, which, it seems worthy of mention, was relieved by chloretone when everything else had proved useless.

This picture comprises four points worthy of remark, three of which are uncommon and one of which is distinctly rare. The first of them, nystagmus, has been observed on many occasions, but when observed it has generally seemed deserving of special mention. Thus Ross and Bury[1] refer to cases as having been recorded by Hadden, Oppenheim, Vierordt, Thomsen, and the authors. On the other hand, Gowers and Taylor[2] state that "slight nystagmus is common," and Purves Stewart[3] says that "on extreme lateral deviation it is not uncommon." Judson Bury,[4] however, declares it to be rare, and the disease in question is certainly not included in the ordinary text-books as one of those in which nystagmus is often met with. This omission is to be regretted, because it might very easily provide a pitfall for the unwary.

Of the retention of power in the supinator longus only this need be said, that it is generally referred to as a valuable point in the differential diagnosis between neuritis due to lead and that due to alcohol, so that the fact that the muscle is not infrequently spared in the latter is apt to be lost sight of. Nevertheless Gowers,[5] Judson Bury,[6] and Williamson,[7] all refer to the occasional occurrence of the phenomenon, though in a manner, it must be admitted, which does not serve to attract the amount of attention which seems to be demanded by an important exception to a well-recognised rule.

The involvement of the bladder and rectum at the outset was probably due to the psychical state, but this explanation does not suffice to account for the persistence of the rectal incontinence long after the control of the bladder had been regained. When I saw the patient, though the intelligence was impaired in the manner above stated, the general note was one of exaltation, which does not usually go with dirty habits. Gowers[1] and Judson Bury[2] are both of opinion that such incontinence is so rare as to be highly suggestive of involvement of the cord. If the cord was involved in this case, there was no other evidence of the fact.

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[1] *A Treatise on Peripheral Neuritis*, p. 155.

[2] *Diseases of the Nervous System*, Vol. I., p. 157.

[3] *A Manual of Medicine* (Allchin), Vol. III., p. 256.

[4] *System of Medicine* (Allbutt), Vol. VI., p. 678.

[5] *Diseases of Nervous System.*

[6] Allbutt's *System of Medicine.*

[7] *Encyclopædia Medica.*

The absence of foot-drop is so striking a departure from the orderly course of an alcoholic peripheral neuritis that one might be forgiven for declining, in its absence, to admit the diagnosis. It was in this case, however, so difficult to escape from the conclusion to which the history and positive symptoms seemed inevitably to point, that I searched the literature to see if anything of a similar kind had ever been recorded. There is one case, and, so far as I could ascertain, one case only, which appears to resemble that above described in its more important features. This is one narrated by Dr. Leslie H. Jones[3] of a lady who suffered an alcoholic neuritis of the ordinary type, who recovered and remained well for some time, but who unfortunately relapsed into her former habits and had a second attack. In this second attack the hands and arms were very severely affected, but the dropping of the feet was not nearly so marked as on the first occasion.

This, then, may be regarded as confirmatory evidence of the fact suggested by the case I have related, namely, that in a second attack of alcoholic neuritis not only may the legs be involved to quite a minor extent, but that they may actually escape altogether. It is as if the disease conferred an immunity from a second attack upon those nerves which have once been severely affected.

The second case to which I wish to refer, as instancing a complete absence of any of the recognised causes of peripheral neuritis of the alcoholic type in a patient suffering from many of the symptoms of the disease, is one which is now in the German Hospital under the care of my colleague, Dr. Parkes

[1] *Diseases of Nervous System.*

[2] Allbutt's *System of Medicine.*

[3] *British Medical Journal*, April 13, 1901.

Weber, to whose courtesy and ready assistance I am indebted for the particulars and for the permission to publish them.

The patient is an Englishman of 46, a baker by trade, who, though he admits that the exigencies of doing business compelled him to take more drinks than were altogether desirable four or five years ago, has nevertheless been a complete teetotaller for over two years. He is very intelligent, and his manner is such as to carry conviction that his statements are trustworthy. The question of alcohol may, therefore, be dismissed. The bakery in which he works is above ground, and he is not exposed to any fumes from the oven. He has not had influenza or other serious disease; he has never resided out of England; he has not been taking any medicines, and has never, to his knowledge, had syphilis. He is unable to offer any suggestions as to the cause of his present trouble, beyond the fact that he has always used his legs a good deal, has been exposed to all weathers, and has not been as particular in changing wet clothes as perhaps he ought to have been. It is not, however, suggested that the present attack is the direct outcome of such exposure or such carelessness. The cause is, in fact, altogether obscure.

So far as the symptoms are concerned, they conform to the ordinary alcoholic type. On admission, he complained of foot-drop, pains in the calves, and stiffness in the fingers, all of which, though of gradual onset, had shown themselves strongly during the previous week. He was admitted on the 1st of January, and had been at work up to Christmas Eve. Since his admission the loss of power had extended to the hands, and, at the time of writing, he has complete double foot- and wrist-drop, with impaired sensibility to touch in both feet. There are no ocular phenomena, and the sphincters are not involved. The supinator longus also is unaffected. The mind is, and has all along been, perfectly clear.

The interest and, if I may say so, the importance of this case resides in the possibility, which so seldom presents itself, of positively excluding not only both alcohol and arsenic, but every other factor to which the disease is usually attributed; and it suggests caution in assuming, as is sometimes done, the existence of such factors where no definite history of them is to be obtained.

THE AFTER-TREATMENT OF EMPYEMA IN INFANTS.

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WHEN an empyema has been diagnosed, and an operation for its relief has been performed, the treatment of the patient has only just begun. The good or bad results that follow upon an empyema are less dependent upon the operation than upon the after-treatment.

The mortality of empyema is high amongst patients under two years of age. At this age the disease has peculiarities, some of which tell in favour of a quick and good recovery, whilst others tell against it. They all add special features, which must be borne in mind in carrying out the treatment. In babyhood a pneumococcal origin is very much commoner than in adult life; in fact it may be said that the vast majority of empyemata in infants are caused by the pneumococcus. At this age also reëxpansion of the lung takes place much more readily and more rapidly than in later life. Both these peculiarities make the prognosis for infant patients better than is the case in adults. But another peculiarity, namely, the greater liability to complications, renders the disease more serious than in adults. These facts are well recognised, but the application of them as they affect the after-treatment is not so fully realised as it should be.

Before proceeding to deal with special points, it will be well to consider briefly the treatment of an uncomplicated case of empyema in an infant from the time that an operation has been performed and a drainage-tube inserted. After the operation there is, sometimes, a certain amount of respiratory embarrassment from the sudden alteration of the thoracic conditions. Dyspnœa of a more or less marked character and slight blueness of the lips, not present before, may be noticed.

This is best met by inhalation of oxygen ; a cylinder of oxygen should be placed by the side of the bed and an india-rubber tube attached to it, so that the nurse can direct a gentle stream of gas into the patient's face. Much may in this way be done to tide over the period before the circulation becomes accustomed to the new respiratory conditions, and to prevent the heart from becoming exhausted. This method should be resorted to whenever the breathing becomes difficult or there are signs of cyanosis at an early stage. At a later period symptoms of this kind are of graver import, for they generally mean that bronchopneumonia, the commonest and most serious complication, is present ; then, although oxygen may be tried, it will probably not do the same amount of good.

If the child seems to be able to breathe more easily sitting up, it should be nursed as much as possible in this position. It is most important that the expansion of the chest and the descent of the diaphragm should not be impeded by tight bandages or heavy clothes round the chest or by a binder round the abdomen. The child should be clothed in some loose flannel garment. The cot should be placed in a situation away from draughts, but should not be surrounded by a tent or anything that will render the air breathed stagnant, or prevent the proper access of fresh air to the patient. The child should be got out of the sick-room as soon as possible ; and should the weather and season be favourable, it will often be possible to do this at the end of a week or ten days. A little later a change to the seaside or the country is of the greatest value in hastening recovery.

The dressings should be changed frequently if there is much discharge, and the same scrupulous care must be taken to secure asepsis during the dressing as in dealing with a sterile wound. It is most important that fresh septic infection should not be engrafted on to that already present.* The majority of empyemata in infants being pneumococcic in origin, the addition of a staphylococcic or streptococcic infection will involve a much more serious condition.

When the dressing is changed, after the tube has been removed, the patient should be turned well over on to the affected side so as to allow any pus to run out which may have collected at the back of the cavity and which has not been able to find an exit by the tube. The latter must be removed each day and carefully cleaned before being re-inserted; otherwise it may become blocked by flakes of lymph or pieces of pyogenic membrane. With regard to washing out the pleural cavity, in many cases no harm ensues from this procedure; but in not a few, symptoms of serious collapse and even sudden death have resulted from it, even when it has been most carefully carried out. It is better, therefore, to be content with simple drainage of the cavity, especially as in infants the large pieces of pyogenic membrane seen in the empyemata of adults do not form as a rule. The practice of washing out the pleural cavity with antiseptics is particularly objectionable, as some of the fluid is almost certain to remain afterwards in the chest, and, if it be a poisonous antiseptic, it may in an infant cause serious symptoms. The tube itself should be a soft rubber tube of large calibre, and only just long enough to go through the chest-wall. No advantage is gained by having the tube any longer than this; in fact, the drainage is better with a short tube than with a long one. The tube should be looked upon as a means of keeping patent a purely temporary exit for the fluid. It should not be regarded as a means of keeping a cavity open, so that it may heal from the bottom. The cavity of a successfully-treated infantile empyema is obliterated almost entirely by reëxpansion of the lung, and the only part which closes and is healed by granulation is the temporary opening. Since this reëxpansion tends to take place readily and quickly in infancy, the obvious indication is to get rid of the tube at an early date. The same is indicated by the nature of the pleural infection.

The importance of removing the tube early cannot, we feel, be too much emphasised; for, although many authorities have insisted upon this point in dealing with the purulent pleural effusions of infants, there is still too little attention paid to it. Much more harm is done by leaving the tube in too long than by taking it out too soon. If a reäccumulation of pus occurs, the wound can easily be reopened, and the tube, if necessary, inserted for a few days longer. On the other hand, when the tube is left in too long, it often gives rise to troublesome complications. Keeping the tube in too long tends to prevent reëxpansion of the lung; also a bottle-shaped sinus, which may be very difficult to cure, not infrequently results. Moreover, the longer the tube is retained the greater is the risk of secondary infection. Sometimes the tube can be left out 48 hours after the operation. Under the most favourable circumstances of all, *i.e.*, when a moderate-sized or small empyema has been operated upon soon after its origin, and when the general condition of the patient is good, the tube can be removed at the end of the second day, and everything should be healed within a week. In a case of average severity, when the circumstances may not be quite so favourable, the tube, should, as a rule, be got rid of before nine days are over; the wound may be plugged with gauze for a few days more, and everything should be healed in about a fortnight.

In estimating the right time to do without the tube, attention must be paid to all the following points :—(1) The length of time during which the empyema has been present before the operation was performed; (2) The character of the pus at the time of the operation and of the discharge afterwards; (3) The size of the empyema ; and (4) The general condition of the patient, especially the degree of anæmia and wasting, the respiratory capacity and crying-power, the cough, the temperature, and the weight. If the empyema is of recent date, the lung will reëxpand more rapidly than when the effusion has remained *in situ* for some time before being let out. If the empyema is a very large one, the lung is the more likely to be extensively collapsed, and will take longer to become inflated again. When the pus is very thick, this is generally associated with chronicity and surrounding adhesions and fibrosis, rendering the obliteration of the cavity slow. When the discharge has become scanty and sero-purulent, or serous, unless there is some obvious contra-indication, the use of the tube can be dispensed with. When an enfeebled baby does not show a good crying-power the respiratory force and the rate of healing of wounds and of inflation of the lung are diminished, and the empyema-cavity takes longer to become obliterated. Such are some of the features to notice in considering when to remove the tube.

In many cases, however, the after-treatment does not resolve itself into the simple questions as to when to leave out the tube, the general hygiene of the patient, &c. Often such difficulties as are associated with judging correctly the time for the removal of the tube are complicated with others of even greater importance. When the progress towards recovery is slow, when fever returns, when the appetite is poor, it is often very hard to ascertain the cause of the non-improvement.

If such a state of affairs exists, there are three questions to be asked, namely: (1) Is there a reäccumulation of pus in the part of the pleural cavity already drained, owing to inefficient drainage? (2) Is there a purulent collection elsewhere? or (3) Has some other complication arisen?

The answers to these questions can only be arrived at by a careful investigation of the physical signs and symptoms. After an empyema has been drained, it is important that the chest should be examined frequently. Careful observation of the physical signs is specially important in infants, as less reliance can be placed upon the temperature than in adults, and the other general symptoms are sometimes misleading. The points to which particular attention must be directed are the position of the heart, the state of the opposite lung and pleura, and the physical signs on the diseased side. If the heart is displaced before the operation, it should quickly return to its proper position after drainage has been established. If it does not do so, there must be some reason for its non-return; and the fact of its continued displacement may be most helpful in indicating the presence of some other collection of pus. The cardiac sounds and impulse should be carefully noted in every case, for only by such means will the recognition of a slight variation from the normal enable the observer to be put upon the track of making that most difficult of diagnoses, the diagnosis of purulent pericarditis complicating empyema. This variety of pericarditis is hardly ever accompanied by a friction-sound. It is the lack of examination, too, and occasionally of experience in examining, that accounts for the frequent oversight of an empyema on the opposite side of the chest; for it is by no means easy to appreciate the existence of an empyema on one side of the chest of an infant, when the physical signs on that side have to be compared with the opposite chest which is already diseased and abnormal; and it is largely by frequent comparisons of percussion-resonance on the two sides, in cases where one side is healthy, that the recognition of a second empyema becomes possible. But most important of all, and perhaps least often investigated, are the physical signs on the diseased side. Roughly speaking the signs over the empyema-cavity which has been opened are those of a localized pneumothorax, only with, as a rule, this difference, that the percussion-resonance is more likely to be impaired than to be hyper-resonant. The probable explanation of this lies in the thickening of the pleura, which usually occurs from swelling of its substance or fibrinous exudate on its surface. Now if these signs vary much from those which usually occur under the circumstances, especially if the amount of impairment of percussion-resonance becomes progressively and markedly greater, they point to a reäccumulation of pus in the original cavity, or to a second pocket of pus. Which of these two complications has occurred can sometimes be told by the position of the dulness.

With regard to the treatment of these complications when they have arisen, it is most important, so long as drainage is required, that the opening into the chest should be wide. If in spite of careful attention the wound gets narrowed, the drainage ceases to be free and the pus tends to be retained, there should be no hesitation in at once enlarging the wound and, if necessary, removing a piece of rib to secure free drainage. When symptoms and signs of retained pus still persist, although the cavity is apparently draining quite freely, search must be made for a second pocket of pus encysted in some cavity other than that opened. A very good plan is to pass a catheter into the wound and push it in various directions until the pus is found. When there is an empyema on the opposite side of the chest, it is best to be content with simple aspiration of the pus for a day or so, until there has been time for a certain amount of reëxpansion of the lung on the side first affected. Later on a second operation can be performed.

A troublesome complication that sometimes occurs after empyema is the presence of a persistent sinus. This may be due to several different causes; thus it may be due to non-expansion of the lung; this, though the commonest cause among adults, is a rare one with infants, in whom the lung seldom fails to expand. In the absence of this cause, the presence of dead bone should be suspected, and looked for by carefully exploring the sinus with a probe. A portion of the rib is often found to be necrosed, the necrosis being most commonly along the lower border of the rib above the sinus. When this is the case, the whole of the necrosed portion of rib must be removed before the sinus can be expected to heal. Inadequate drainage from the chest is not an uncommon cause of persistent sinus. When the discharge from the sinus is intermittent, that is to say, when the wound keeps healing up and breaking down again, this cause must especially be suspected. The treatment, of course, is to reopen the wound freely, and drain all the pockets of pus that are found, or pack them with gauze and allow them to granulate up from the bottom. Other and rarer causes of persistent sinus are tubercular disease of the pleura and caries of the spine, to which the empyema may have been secondary.

In considering the treatment of the local condition, the general treatment of the patient must not be overlooked. Stimulants are often valuable, and should be exhibited when indicated. Free stimulation should be employed when secondary bronchitis or bronchopneumonia arises and endangers the life of the patient. Belladonna also may be used with advantage under such circumstances. General tonics may be useful in some cases. Careful supervision of the diet is also necessary to combat the tendency to gastric or intestinal catarrh, which so often arises. These small patients are particularly liable to contract diarrhœa, so that careful attention should be paid to the prophylactic measures against this complaint, especially in an infant under a year old and when the empyema occurs during the hot summer months. It is wise, if the baby is being fed on the breast, not to let the occurrence of an empyema enforce weaning.

In conclusion, emphasis must be laid upon the importance of paying great attention to the general symptoms. This is perhaps less frequently omitted than the investigation of the physical signs. The continuance of fever, or the non-return of the appetite, should at once sound the alarm and warn us to be on the look-out for complications.

It is unnecessary to do more than mention that a routine examination of the weight, of the rate and quality of the pulse, of the respiratory functions (noting especially any cyanosis or recession of the chest-wall), of the abdomen, and of the functions of its contents, are most important aids in diagnosing the existence of the complications already mentioned, while on the other hand they are often the only means of indicating to us at an early date the onset of some other complication such as suppurative meningitis or pyæmia.

THE PRESENT POSITION OF OUR KNOWLEDGE OF DISEASES OF THE PANCREAS.

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DURING the last twelve years our knowledge of the physiology and pathology of the pancreas has increased rapidly. In 1888 the experiments of Martinotti, and in 1889 the well-known work of Von Mering and Minkowski, laid the foundation of much of our present acquaintance with the diseases and functions of this gland. It must be admitted, however, that even at the present time our knowledge is fragmentary, and that a great field yet lies before the investigator and the clinician.

I propose in the following brief *résumé* of the diseases of the pancreas to deal with the subject under the following headings:—

I.—Experimental Work on the Pancreas.
II.—The Signs and Symptoms of Pancreatic Disease.
III.—Cysts of the Pancreas.
IV.—Calculus.
V.—Pancreatitis—acute, subacute, and chronic.

I.—EXPERIMENTAL WORK ON THE PANCREAS.

Almost the whole of our knowledge upon this point is derived from the investigations of Von Mering and Minkowski.

a. Total extirpation of the pancreas produces, in addition to certain digestive disturbances, all the symptoms of diabetes—glycosuria, polyuria, polyphagia, and gradual wasting. Unless the entire gland is removed, diabetes does not occur as a permanent disease.

b. Partial extirpation of the pancreas never results in permanent diabetes. Even if only the tail of the gland is left, diabetes is not established. Hédon has removed portions of the gland at three successive operations at long intervals. Diabetes did not occur until all the gland had been extirpated. It is clear, therefore, that pancreatic diabetes is the result of the absence from the body, not of the pancreatic juice, but of some secretion analogous to those known as "internal" in the thyroid and other glands; and that the want of this secretion cannot be vicariously supplied by any other structure or gland, when the pancreas is removed piecemeal. If a very small portion only of the gland remains, and if this undergoes a process of gradual atrophy, concomitantly a gradual onset of diabetes is observed.

c. Ligature of the duct of the pancreas gives rise to a chronic interstitial inflammation in the gland. The duct does not dilate behind the ligature. It has been repeatedly shown that after this operation diabetes does not occur. Ssobolew has observed that the islands of Langerhans are unaffected by the chronic inflammatory process.

d. Subcutaneous transplantation of the pancreas.—In dogs the descending portion of the pancreas is rendered mobile, its blood-vessels being left intact, and is grafted into the subcutaneous cellular tissue of the abdominal wall; the secretion is passed through a fistula. If after a time the vasculo-nervous pedicle be divided, the graft thrives and continues to secrete for a time, but generally atrophies. If the graft remains active the intra-abdominal portion of the gland can be removed without causing diabetes; but if, after removal of the intra-abdominal portion, and after an interval of a few days, the graft be removed, glycosuria immediately develops.

The relation of diabetes to the condition of the pancreas has attracted attention, since Cawley in 1788 first recorded the existence of calculi in the duct of an atrophied pancreas in a case of diabetes. In 1877 Lancereaux emphasised the connection between diabetes and disease of the pancreas. He and his pupil Lapierre showed that in *diabète maigre,* a form of diabetes characterised by brusqueness of onset, by rapidity of course, and by the serious character of the symptoms, an atrophy or other profound alteration in the pancreas could be found. Lancereaux, in his *Traité des Maladies du Foie et du Pancréas,* described the following conditions as having been found in cases of *diabète maigre* :—

(1) Aplasia of the pancreas — a simple diminution in the glandular elements, without alteration in their character;

(2) Atrophy, with sclerosis and fatty changes;
(3) Atrophy consecutive to inflammation and obliteration of the ducts; and
(4) Tumours, notably cancer of the head of the gland causing duct-obstruction.

Embedded in the substance of the pancreas are certain groups of polygonal cells, arranged in columns, known as the *islands of Langerhans*. It has been suggested by Laguesse, Schäfer, Diamare, and others that these islands are concerned in the production of the "internal secretion" of the pancreas. If the suppression of the internal secretion of the gland causes diabetes, and if the secretion is the product of these islands, then in a case of pancreatic diabetes we should expect these islands to be the seat of some pathological condition. (The islands of Langerhans are probably the morphological representatives of the primitive pancreas of the cyclostomata.)

Opie, of Baltimore, has, in a series of remarkably interesting papers, given the following explanation of the cause of pancreatic diabetes in man. He described two forms of interstitial pancreatitis:—

(*a*) *Interlobular pancreatitis*, in which the inflammatory process is localised chiefly at the periphery of the lobule, and implicates the islands of Langerhans only when the sclerotic process has reached a very advanced stage. When pancreatitis has followed obstruction of the ducts, the islands long remain unaltered, though embedded in dense scar-like tissue.

(*b*) *Interacinar pancreatitis*, in which the process is diffuse, invading the lobules and separating individual acini. The inflammatory change invades the islands of Langerhans.

In one of 11 cases of interlobular pancreatitis, diabetes of mild intensity occurred. The sclerosis in this case was far advanced and affected the islands of Langerhans. In 2 out of 3 cases of interacinar pancreatitis, diabetes was present. The third case was associated with a condition, hæmochromatosis, which at a later stage is associated with diabetes, the result of a pancreatic lesion.

In a later paper, Opie gives a full and lucid account of a case of diabetes, in which the causal relation of a lesion of the islands of Langerhans to the disease is demonstrable.

He concludes that destruction of the pancreas in animals and in man is accompanied by diabetes; in the case he describes the destruction of the islands of Langerhans was accompanied by this disease. Since diabetes is absent when, as the result of duct-obstruction, the secreting portion of the gland undergoes great alteration, though the islands are spared, the conclusion is justified that it is those structures which influence carbohydrate metabolism. What has been learned concerning the relation of the pancreas to diabetes is the relation of the islands of Langerhans to this disease. Weichselbaum and Stangl, in an investigation of the pancreatic changes in 18 cases of diabetes, found atrophy of the parenchyma in 17. The islands of Langerhans were fewer in number and much smaller than normal. These observers conclude that the changes described are not due to pancreatitis or granular atrophy, but to a genuine atrophy, from some unknown cause, especially affecting the islands of Langerhans.

II.—THE SPECIAL SIGNS AND SYMPTOMS OF PANCREATIC DISEASE.

In addition to glycosuria, which is almost certainly due to an atrophy of, or alteration in, the islands of Langerhans, certain other signs and symptoms are often to be attributed to lesions of the pancreas. Of these may be mentioned:—

1. Hæmorrhage.
2. Fat-necrosis.
3. Altered character of the stools.
4. Alteration in the constituents of the urine.
5. Wasting.

1. *Hæmorrhage.*—The association of hæmorrhage with diseases of the pancreas has been well recognised since Fitz's classical paper upon "Acute Hæmorrhagic Pancreatitis" in 1889. Hæmorrhage may occur spontaneously into the substance of the gland, and may even prove fatal; or it may occur concomitantly with acute, subacute, or chronic inflammation. All these forms of inflammation may, however, occur without any evidence of local bleeding. Experimentally hæmorrhage may be produced by the injection into the duct, of Wirsung of the Bacillus diphtheriæ, or of the Bacillus

pyocyaneus. Mayo Robson thus arranges the facts regarding the association of hæmorrhage and pancreatic disease :—

α. In certain diseases of the pancreas there is a general hæmorrhagic tendency, which is much intensified by the presence of jaundice.

β. Hæmorrhage may apparently occur in the pancreas unassociated with inflammation, or with jaundice, or with a general hæmorrhagic tendency.

γ. Acute, subacute, and chronic pancreatitis can, and do, frequently occur without hæmorrhage.

δ. Acute pancreatitis is so frequently associated with hæmorrhage into the substance of the gland as to establish the need for the name "hæmorrhagic pancreatitis" as a variety of acute inflammation. In one case of acute inflammation going on to gangrene of the pancreas, I have observed the occurrence in the subcutaneous tissue of the body and limbs of large hæmorrhages.

As will be seen in discussing the question of fat-necrosis, there is, when the fat is split into fatty acids and glycerine, an absorption of the latter. It is possible that the glycerine may to some extent be responsible for the hæmorrhagic tendency, for numerous clinical observations go to show that glycerine (as, for example, when joints or other tubercular affections are treated by the injection of an emulsion of iodoform and glycerine) is capable of causing local hæmorrhage and occasionally hæmaturia.

2. *Fat-necrosis.*—This extremely inappropriate name was suggested by Balser in 1882, when he gave the earliest description of the phenomenon. The condition is not one of necrosis, but of saponification. The fat so affected shows, on section, white patches, irregularly distributed throughout its substance. These patches may be found in the abdominal wall, and in any part of the fat within the abdomen ; they have also been observed in the fat beneath the pericardium. It is said that fat-necrosis has been observed apart from a demonstrable lesion of the pancreas, but such instances form but a small proportion of the whole. In by far the greater number an obvious affection of the pancreas, acute inflammation of the hæmorrhagic or necrotic type has been found. Fat-necrosis is caused by the splitting up of the fat into fatty acids and

glycerine; the latter is absorbed, the former combines with the calcium salts and produces the white soapy patches characteristic of the disease.

Langerhans, who was one of the earliest investigators, attributes the fat-necrosis to the escape of the fat-splitting ferment of the pancreatic juice. The affection of the fat immediately adjacent to the pancreas is due to contact with the juice as soon as it escapes; fat lying at a distance from the gland is probably affected by juice conveyed to it by the lymphatics.

Opie's investigations on this question have resulted in the following conclusions:—

α. The condition essential to the production of focal fat-necrosis is the penetration of the fat-splitting ferment of the pancreas into living adipose tissue.

β. By causing the entire secretion of the pancreas in cats to penetrate into the tissue surrounding the organ, wide diffusion takes place, and necrosis results, not only in the abdominal fat, but in that of the pericardium and subcutaneous tissue as well, thus reproducing the widespread distribution of the lesion occasionally seen in man and in the lower animals.

γ. Lesions of the pancreas associated with focal fat-necrosis are such as to permit the diffusion of the pancreatic ferment into the surrounding tissue.

3. *Altered Condition of the Stools.*—Claude Bernard, and Dr. Walker, of Peterborough, have shown that the absence of the pancreatic secretion from the intestine results in the passage of clay-coloured stools. The coloration of the fæces is not due to the bile-pigments, which are all absorbable, but to a pigment which is insoluble, and which results from the action of the pancreatic juice upon some of the colouring matters of the bile. The absence either of the bile or of the pancreatic juice results, therefore, in the passage of unpigmented fæces.

It is important to remember that the pancreas has normally two ducts, the duct of Wirsung and the duct of Santorini, which open separately into the duodenum. The blockage of one of these alone would not suffice to check the entrance of all the secretion of the gland into the bowel. Both ducts must be blocked, or the gland so altered that no juice is secreted by it.

The presence in excess of *undigested muscle-fibre* in the stools is also an indication of pancreatic inadequacy, as pointed out by Küster and others.

The appearance of *fat in excess* in the fæces was first observed in connection with pancreatic disease by Kuntzmann, in 1820.

The altered conditions of the fæces that may be associated with pancreatic disease are, therefore, absence of pigmentation, the presence of undigested muscle-fibre in excess, and the presence of fat in excess. The stools are often bulky, much of the food being undigested, and passing unabsorbed through the intestine.

4. *Alteration in the Constituents of the Urine.*—A reduction in the amount of ethereal sulphates in the urine has been thought to be a possible sign of pancreatic disease, for the following reasons:—The production in the intestine of the substances which are, finally, largely excreted in the urine as so-called ethereal sulphates, is dependent upon the putrefaction of protein, and this process is due to bacterial activity. Bacteria are able to break down native albumen in only relatively small amounts, while they readily decompose comparatively large amounts of the products of proteolytic digestion. If, therefore, there were little or no proteolytic digestion going on in the intestine, as is the case in severe diseases of the pancreas, the products of bacterial activity (indol, skatol, &c.) would be largely decreased, and there would consequently be a reduction in the ethereal sulphates of the urine. Edsall concludes a critical examination of the literature of this subject with the following statement. If the test as to the presence of ethereal sulphates is negative, the result deserves little consideration; if, on the other hand, the ethereal sulphates are found much reduced, the other factors in the case should be considered in connection with this observation. If there is diarrhœa or gastric hyperchlorhydria, or if the patient is being fed on milk alone, or any other diet which very largely reduces bacterial activity, a low ratio of the ethereal sulphates cannot be considered of great importance, nor can much stress be laid on a low absolute value of the ethereal sulphates, if the values for the inorganic sulphates be coincidently low. If, however, these

factors be not present, and particularly if, with suspicion of pancreatic disease, factors which usually cause an increase of the ethereal sulphates be present, and yet the values be found low, the test would at present appear to be of distinct practical importance in diagnosis. Constipation, gastric hypo-acidity or anacidity, icterus, grave anæmia, and cachexia are all very likely to cause an increase in the relative or absolute values of the ethereal sulphates.

5. *Wasting.* — The rapidity with which the body-weight declines in cases of pancreatic disease can only be matched in malignant disease. The reason for this is obvious when we remember that the brunt of the work of digestion is borne by the pancreatic secretion. If this secretion is absent the greater part of the food passes unaltered through the intestinal canal.

PANCREATIC CYSTS.

The study of cystic diseases of the pancreas is one beset with difficulties, for our knowledge of their pathology is slight, and clinical observations are comparatively few and imperfect. The following is a brief epitome of our present knowledge.

Pancreatic cysts are of the following varieties :—

1. Retention-cysts.
2. Proliferation-cysts { cystic adenoma. / cystic epithelioma.
3. Hydatid disease.
4. Congenital cystic disease.
5. Hæmorrhagic cysts.
6. Pseudo-cysts.

From the pathological point of view the inclusion of the last form is not permissible. As however, the clinical distinction between these and the true forms is often impossible, it seems best to include a description of them.

1. *Retention-cysts.*—Virchow (*Die krankhaften Geschwülste,* 1863, Vol. I., p. 276) described two forms of retention-cyst of the pancreas. In the one the whole duct is widened, and a "rosary-like" dilatation occurs. In the other the duct is blocked at its outlet, and becomes distended into a cyst, which may reach the size of a fist, and may contain

mucoid, hæmorrhagic or calcareous matter. In the case of the smaller cysts there can generally be little or no difficulty in determining their origin; in that of the larger cysts, such as the surgeon is destined to encounter; the differentiation may be difficult or impossible. Virchow himself has expressed his inability to determine the origin of a large cyst attached on the one side to the pancreas, and on the other to the stomach. The blockage in the duct may be due to:—

(*a*) The impaction of a calculus or calculi.
(*b*) Cicatricial stenosis.
(*c*) Pressure upon the duct from without.
(*d*) Dislocation of a part of the gland.

Retention-cysts have been recognised as such during the course of the operation by several observers. In Ludolph's case (*Inaug. Dissert.*, Bonn, 1890) the cyst was excised and the opening of the duct clearly seen. Hagenbach found two dilated radicles of the main canal, opening into the cyst. Dixon (*Medical Record*, March, 1884) found the duct opening into the larger of two cysts. Richardson's case was considered by him to be a cystic dilatation of the duct. As it is rarely possible to make an exhaustive inspection of the parts during an operation, it is perhaps remarkable that so many observations have been recorded.

On the post-mortem table specimens have been recognised by Virchow, Klebs, Gould, and many others. Virchow applied the term "ranula pancreatica" to a general distension of the whole duct. When numerous small engorgements of the minute ducts are present Klebs suggested the name "Acne pancreatica."

2. *Proliferation-cysts* may be either simple or malignant. The exact differentiation is not infrequently difficult, and is at times impossible from the pathological point of view. One has in some cases to await the course of events after the removal of a cystic tumour before deciding as to the class in which to include it. Thus Fitz, of Boston, in relating his case (*Amer. Journ. of Med. Sci.*, August, 1900), says: "The tumour, from its histological appearances, is to be regarded as essentially a multilocular cystoma, but on the border line between a proliferating cystoma or cystadenoma and a cystomatous carcinoma, a distinction which the

subsequent history of the patient may be expected to make clear."

3. *Hydatid-cysts* of the pancreas are extremely rare. Masseron *(Thèse de Paris,* 1881) was able to collect the records of only five cases, and these were first recognised on the post-mortem table. Graham, of Sydney (*Hydatid Disease in its Clinical Aspects,* 1891), writes: The hydatid is sometimes found in the pancreas. I have observed it as a cyst about three inches in diameter replacing the head of the organ." Tricomi states, without giving references, that seven cases have been recorded.

4. *Congenital cystic disease,* similar to the congenital cystic disease of the liver and kidneys, has been observed on rare occasions. Dr. Pye-Smith recorded (*Path. Soc. Trans.,* 1885, p. 17) a case of cyst of the cerebellum, with numerous small cysts in the pancreas and kidneys. The patient was a man aged 27. Richardson has recorded a cyst of the pancreas occurring in a child 14 months old; and in one case, related by Shattuck, a tumour had been noticed since birth.

5. *Hæmorrhagic cysts.*—That bleeding may occur into the substance of the pancreas as the outcome of acute or chronic inflammation is well proven. As to the influence of these or other hæmorrhages, traumatic or spontaneous, upon the building up of a cyst, no positive opinion can be expressed. Hagenbach distinguishes between hæmatoma, in which bleeding occurs into preëxisting cysts, and apoplectic cysts resulting from hæmorrhage into softened, degenerate gland-substance. In favour of the hæmorrhagic origin of cysts are the writings of Kühnast, Schröder, and Friedreich.

Gussenbauer conjectured that the origin of the cyst in the case operated upon by him was due to a central hæmatoma of the pancreas. In discussing the case Senn remarks: "This assumption lacks demonstration, and it is just as logical to assume that the cyst originated in the usual way from an obstruction, and that the blood in the cyst contents was an accidental product"—a comment with which Körte seems in accord.

6. *Pseudo-cysts.*—Körte has proposed the term "pseudo-cysts" for certain fluid tumours found in more or less close

proximity to the pancreas, but not originating in the substance of the gland. The accurate distinction of a true pancreatic cyst from a pseudo-cyst is not always possible during life, for a cyst primarily peri-pancreatic may originate in a trauma, which implicates the gland at the same time, so that the pancreatic juice may in small quantity escape into the pseudo-cyst and make its mimicry of a true cyst so complete that a distinction is impossible. I am indeed strongly inclined to believe that many cases of so-called pancreatic cysts, especially those of traumatic origin, are in reality peri-pancreatic or pseudo-cystic effusions into the lesser cavity of the peritoneum, localised extravasations of blood, and so forth. In some recorded examples the origin of a cyst in the pancreas is purely hypothetical, and in these the tumour may be a "pseudo-cyst" of the pancreas or a true cyst of a neighbouring organ, such as the suprarenal body or the kidney.

The most frequently-occurring form of "pseudo-cyst" is that first described by Mr. Jordan Lloyd (*Brit. Med. Journ.*, November, 1892). In this very important paper attention is drawn to effusions into the lesser peritoneal cavity, as the result of injury to the pancreas, and two cases are fully reported. Mr. Jordan Lloyd writes: "The diagnosis of pancreatic cyst appears to me often to have been made upon insufficient evidence. The fact that a cavity within the abdomen contains pancreatic secretion is no proof whatever that the cavity is within the pancreas; it tells us nothing more than that the cavity is connected with this organ. Neither is the fact that the pancreas can be felt by an examining finger from within an intra-abdominal sac, for if the lesser peritoneal cavity is opened from below by an incision in the transverse meso-colon, a finger passed through this opening enters a large space, at the back of which the pancreas is found. Similarly, too, if the lesser peritoneal cavity is opened by an incision in the great omentum below the stomach. Through either opening the deepest part of this space is found to the left, and cannot be reached by the examining finger."

Mr. Jordan Lloyd summarises his conclusions as follows:—

a. That contusions of the upper part of the abdomen may be followed by the development of a tumour in the epigastric, umbilical, and left hypochondriac regions.

β. That such tumours may be due to fluid accumulations in the lesser peritoneal cavity.

γ. That when the contents of such tumours are found to have the property of rapidly converting starch into sugar, we may assume that the pancreas has been injured.

δ. That many such tumours have been regarded as true "retention-cysts of the pancreas," and that this opinion has been formed upon insufficient evidence.

ε. That the diagnosis of distension of the lesser peritoneal cavity before operation can usually be made by the characteristic shape of the swelling.

ζ. That early median abdominal incision and drainage is the safe and proper treatment.

The following most interesting case is recorded by McPhedran (*Brit. Med. Journ.*, 1897, Vol., I., p. 1400):—

G. A. B., male, 53. In 1891 he had an attack of biliary colic, with well-marked jaundice and pale motions: he had two or three similar attacks every year. His condition became gradually worse; there was almost constantly some epigastric discomfort, indigestion, flatulence. One severe attack of pain lasted three days; the epigastrium was tender, and pain radiated in several directions. He was losing flesh. On examination on a certain date there was an increase in the thickening in the deep part of the epigastrium; three days later a large, smooth cyst-like tumour was found in the epigastrium, extending from the right parasternal line to the left mammary line and down to the umbilicus. The upper boundary was ill-defined; the stomach resonance was above and to the left. A cystic collection in the bursa omentalis was diagnosed, and the abdomen was opened. The cyst was emptied; at the bottom lay the pancreas, irregularly enlarged and firm, but somewhat elastic. The peritoneum over it was smooth and healthy-looking. There was no sign of hæmorrhage anywhere. Five months later a tumour was again found in the epigastrium; this tumour extended down to the level of the anterior superior spinous processes and laterally to the mammary line on the right and to the anterior axillary line on the left. It forced the diaphragm upwards, so that the cardiac impulse was in the fourth intercostal space. The abdomen was again opened and a cyst exposed lying behind the stomach;

the cyst wall was about 2 mm. thick; the fluid was opaque and whitish, and contained many flocculi and fibrinous masses. The fluid was alkaline, and contained albumen, but no digestive ferment. The cavity contracted rapidly, but a fistula persisted; the discharge from this irritated the skin. On examination, the fluid was found to possess marked action on albuminoids, fats, and starches, leaving no doubt as to the presence of pancreatic secretion. The condition causing the repeated attacks of colic lay in the pancreas, and may have been a calculus or a localised inflammatory deposit, causing mechanical obstruction. In the most acute attack the symptoms were those of acute pancreatitis.

This case is epecially interesting from the fact that it is, so far as I know, the only one recorded in which a pseudo-cyst and a true cyst have been observed in the same individual.

Pathological Anatomy.—Cysts of the pancreas are rather more frequent in men than in women. Of 121 cases collected by Körte, 60 were males, 56 females; in the remaining 5 no mention of sex is made.

The youngest patient affected was a child 13 months old, whose case is recorded by Shattuck. Richardson has operated upon a child 14 months old. Stieda's patient was a man of 76.

The cystic tumour may be situated at any part of the gland, from the duodenal margin of the head to the tip of the tail. The body is more commonly affected than the head, and the tendency to implication seems, roughly, to increase as the tail is approached.

In 22 cases collected by Hagenbach the cyst arose in the tail 10 times, 4 times in the head, once in the body, and in 7 the site is not indicated. Nimier gives the following:—21 times in the tail, 6 times on the body, 4 times in the head, once in the head and the body, and in one the whole organ was implicated.

Cysts may be single or multiple, unilocular or multilocular. Two cysts may be simultaneously observed of almost equal size; or, after the healing of one cyst, a second may develop in a distant portion of the gland and necessitate further operation. One small cyst may be found with a large number of smaller ones packed in its walls. The cysts are generally smooth and rounded; they are elastic and rather tense. The inner surface of the cyst-wall is commonly smooth and lined throughout by cylindrical epithelium; trabeculæ or incomplete septa may at times be present, and a "thimbling" of the surface may be seen. Intra-cystic polypoid masses are found in cases of proliferation-cyst. In the great majority of recorded cases blood in greater or less quantity has been observed.

Symptoms and Signs of Pancreatic Cysts.—The symptoms produced by a pancreatic cyst are chiefly referable to the pressure exerted by the tumour upon surrounding viscera. In the earlier stages, while yet the tumour is small, the symptoms are absent, or so trivial as to attract but little attention. Almost imperceptibly the feelings of discomfort, weight, and fulness in the epigastrium increase. Pain is noticed towards the end of, and for some time after, a meal; and vomiting becomes gradually established as a distressing symptom. To these earlier, less pronounced symptoms, Friedreich has given the name "cœliac neuralgia." The pain is generally confined to the upper half of the abdomen, but it may radiate to the back, especially on the left side, and has been said to strike downwards into the testes. The intensity of the pain is liable to wide variation; it may resemble that of "indigestion," or may be acute and almost intolerable, and suggest intestinal obstruction. Vomiting is inconstant and variable, but generally bears a close relationship to the amount of pain suffered. In one case, related by Kocher, pain and vomiting occurred in paroxysmal outbursts every two or three months. Salivation is rarely noticed; the vomiting of clear colourless fluid, "salivatio pancreatica," is recorded by Zielstorff.

The functions of the bowels are generally sluggish: constipation is the rule, but it may give place to diarrhœa at times; a blood-stained liquid motion has been occasionally remarked. In one case, recorded by Indemans, a bloody diarrhœa constantly recurred.

Jaundice is noticed in a small proportion of cases. It depends upon a pressure exerted by the tumour upon the common duct, and is, therefore, associated almost always with a tumour in the head or neck of the pancreas. Körte finds the occurrence of jaundice noted in 9 out of 121 cases.

In the few recorded examples of "giant" cyst the usual concomitant disabilities of largely-distended abdomen are noticed. Dyspnœa is mentioned by Zielstorff, Filipoff, Stapper, and Shattuck.

In almost every case the general health of the patient suffers seriously. Wasting is rapid and continuous. In one of my cases the patient lost three stones of weight in seven weeks. Weakness is very pronounced, and in appearance the patient ages quickly, looking sallow, pinched, shrunken, and profoundly ill. According to Friedreich, wasting is more serious when affections of the bile-passage are associated with disease of the pancreas.

The cystic tumour developing in the pancreas lies behind the posterior layer of peritoneum forming the lesser sac. As the swelling increases it bulges into the lesser sac, and is covered, whatever direction it may take, by this layer of the serous membrane. The tumour is at first placed behind the stomach, but in its gradual enlargement it displaces the viscus. It may pass in one of three directions :—

(*a*) In the great majority of cases the stomach is pushed upwards and to the right, and the transverse colon is pushed downwards, the cyst coming to the surface below the greater curvature of the stomach. In order to reach the cyst through the abdominal wall, the parietal peritoneum, the two layers of peritoneum forming the great omentum, and the posterior layer of peritoneum of the lesser sac have all to be divided. The amount of pressure and displacement to which the stomach and the transverse colon are subject depends entirely upon the bulk of the cyst. The stomach may be jammed tightly under the liver, and the transverse colon may, as recorded by Von Riedel, Salzer, Heinricius, and others, be pushed down as low as the symphysis pubis.

(*b*) In certain cases the enlarging tumour may push its way forward above the upper border of the stomach, and there present. In order to reach the cyst in this position, the following layers of peritoneum must be divided; the parietal, the two layers of the gastro-hepatic omentum, and the posterior layer of the peritoneum of the lesser sac. The stomach is pushed downwards and the liver forced upwards and to the right. The most prominent part of the cyst presses against the anterior abdominal wall. In one case Albert saw a bulging of the cyst through the foramen of Winslow into the greater cavity of the peritoneum. The presenting of the cyst above the lesser curvature of the stomach has been observed in 11 cases (Albert, Indemans, Zielstorff, Riegner, Swain, Hahn, Karewski, Doran, Herman, Finotti). Riegner and Finotti diagnosed the position before operation.

(*c*) If the cyst spring from the lower portion of the head of the pancreas, or from the inferior border of the body or tail, it will be at the lower limit of the lesser sac. As it then increases in size, it will push its way between the layers of the transverse meso-colon, or bulge downwards the inferior layer. If it does the former, the transverse colon will run directly across the front of the cyst; if the latter, the transverse colon and the stomach will both be pressed upwards. Cases in which the transverse meso-colon was invaded are recorded by Zukowski, von Petrykowski, Lardy, Salzer, Riegel, Treves. The tumour then presents as a smooth rounded mass, crossed at or near its middle by the stretched colon. The bulging of the lower layer of the transverse meso-colon is recorded by Hersche, Lindh, and Heinricius. The great omentum may be stretched over the cyst, and thinned by its enlargement, or it may be pushed aside. In the former case, the parietal peritoneum, the great omentum, and the inferior layer of the transverse meso-colon have to be cut to reach the cyst: in the latter, the parietal peritoneum and the inferior layer of the transverse meso-colon only. In one instance, recorded by Bernard Pitts and Shattock, the enteric mesentery invested the front of the cyst. Martin found the descending colon in front of the cyst in his case, and Schwartz the cæcum; in both the tumour was of large size, and occupied the larger part of the abdomen.

Diagnosis.—The signs and symptoms warranting a diagnosis of pancreatic cyst are as follows:—A patient ill, with indefinite symptoms of epigastric uneasiness, weight, pain, and occasional vomiting, begins to lose weight, and on examination a swelling of the upper part of the abdomen is discovered; or, a patient suffers an injury of some severity to the upper part of the abdomen—the kick of a horse, a heavy blow, or, while he is lying on the ground, the abdomen is trampled upon—within a brief period of days or weeks a tumour forms, with or without symptoms, in the epigastric region. As the tumour enlarges, the symptoms become more acute, or, less commonly, remain stationary. Jaundice, intestinal hæmorrhages, or diarrhœa may be observed. The tumour which forms lies generally in the epigastric region, with its most prominent aspect at or near the middle line, or between the mid-line and the left costal margin. The tumour is rounded, generally smooth, and elastic or fluctuating. It may vary in size from time to time. It is dull on percussion in the centre; but above it percussion demonstrates the stomach-resonance, and below, that due to the transverse colon. Inflation of these viscera, as first suggested by Gussenbauer, increases the area of their tympany and lessens the central dull area of the tumour. The tumour is fixed; the skin over it smooth and unwrinkled.

In the urine sugar may be found; in the fæces an excess of fat and of undigested muscle fibre.

Treatment.—The operative treatment of pancreatic cysts is limited to three methods:—

1. Aspiration.
2. Evacuation and drainage, the cyst being stitched to the abdominal wall.
3. Extirpation, partial or complete.

Evacuation and drainage is, as a rule, the most successful form of treatment. A pancreatic fistula may, however, remain for months.

IV.—PANCREATIC CALCULUS.

The occurrence of stone in the pancreas was first recorded by Graaf in 1667. Morgagni in 1765 and, in England, Cawley in 1788, refer to instances observed by themselves. In 1883 Johnston collected the notes of 35 recorded cases. The fullest account was given in 1886 by Giudiceandrea and was based upon 48 recorded cases and two observed by the author.

The stones are generally white, greyish-white or yellowish-white in colour, and rounded, ovoid, or elongated like a date-stone. They are found in all parts of the duct of the pancreas, though much more frequently in the head; in the tail of the gland they are rarely seen. The calculi may be branched like coral, the trunk of the stone lying in the main duct and its offshoots in the secondary ducts. At times the canal of Wirsung is found packed with a coarse mortar-like material, or with sand or fine rounded pellets. The largest stone, measuring two and a half inches by half-an-inch, was seen by Schupmann. Calculi may be single or multiple; one of the most striking examples of the latter is in the Museum of the Royal College of Surgeons of England (specimen 2834, Curnow's case). As many as 300 stones have been found in one case. The stones are chiefly composed of phosphorus and carbon salts. One case of oxalate-of-lime stone is reported by Shattock.

Symptoms.—So few carefully observed cases of pancreatic lithiasis have been recorded that it is impossible to give any accurate portrayal of the symptoms. In the majority of examples pain, or at the least a sense of great discomfort, has been felt in the upper part of the abdomen, generally at or near the middle line; rarely, as in Minnich's case, under the left costal margin. The pain frequently comes in sharp, colicky attacks, similar to, but less severe than, those due to gall-stones. A sense of soreness or stiffness is noticed for a day or two after the attack. When the pain is at its height, vomiting, hiccough, rigors, cold sweats, or collapse may be noticed. After the attack some fragments of stone may be found in the motions. (Minnich, Leichtenstern.) Diabetes has been often observed. Hausemann, in 72 cases of diabetes associated with pancreatic disturbances, found that in 12 stone was present. Osler in 70 cases of lithiasis observed that diabetes was recorded 24 times. The probability is that the onset of diabetes is due to the chronic inflammatory changes instituted in the gland by the calculus, but it must be remembered that such inflammatory alterations produce in the gland and its duct precisely those changes which are most likely to cause the deposit of calculous material. Fatty stools have been occasionally observed, and in one case Lichtheim saw an abundance of fat-crystals in the fæces. Rarely jaundice may be caused, as related by Giudiceandrea and Pearce Gould by the pressure of the stone upon the common duct. Lancereaux states that in each attack of pancreatic colic a temporary diabetes is produced by an inhibition of the secretion, analogous to the inhibition observed when one ureter is obstructed by a calculus. The cardinal symptoms of pancreatic calculus, then, are:—pain, colicky in character, similar to, but of lesser intensity than, gall-stone colic, and generally most intense in the epigastrium; pain or tenderness, and occasionally a feeling of resistance over the pancreas, the passage of fragments of stone, marked bodily wasting, fat-diarrhœa; and in the later stages, diabetes. It is rarely that all these are seen in association.

Diagnosis.—Claessen in 1842 first suggested the possibility of a correct diagnosis of pancreatic calculus being made. Friedreich, though he considered the occurrence of pancreatic colic not unlikely, wrote: "The diagnosis of pancreatic calculus is impossible." Material for a diagnosis was afforded by the cases of Lichtheim, Minnich, Körte, and others. In Minnich's cases the pain began as a heavy pressure in the epigastrium and along the left costal margin; as it increased in intensity it passed round the left side and backwards to the spine. The patient, who had previously suffered from hepatic colic, remarked that the character of the two pains was identical. Körte has related a very interesting and remarkable case. The patient suffered from biliary colic for which the abdomen was opened; a large gall-stone was removed from the gall-bladder, which was drained by a tube. While bile was still being discharged the patient suffered from what he took to be a recurrence of his attacks of biliary colic. The patient died, and a tumour in the head of the pancreas, which had been diagnosed as malignant at the time of the operation, was found to be an abscess with large concretions within it. Kummell records an almost exactly similar case in which cholecystotomy had been performed; pain recurred and at the necropsy a large soft calculus was found in the canal of Wirsung. That pancreatic colic is associated with the passage of stones down the duct seems clearly to be proved by the case observed at different times by Minnich and Holzmann, for it was only after each attack that fragments of stone were found in the motions.

The presence of sugar in the urine may be observed at intervals. Holzmann found it intermittently in his case. While the same patient was under Minnich's observation at an earlier date no sugar was found, though regularly sought. It is probable that it is only when advanced interstitial inflammation of the interacinar type is present, and the islands of Langerhans are therefore affected, that glycosuria is present. Caparelli records the case of a woman who developed, after many attacks of acute epigastric colic, an abscess above the umbilicus: the abscess burst and discharged some pus and gritty material. Through the fistula, which persisted for six years, many small stones, over 100 in all, were expelled. After the spontaneous closure of the fistula diabetes developed, and the patient died. Lancereaux's statement that in each attack of colic there is a temporary glycosura is not borne out by the history of the case below recorded. Very careful and continuous examinations of the urine were made, but sugar was never once discovered.

The following case, recorded by me in the *Lancet*, August, 1902, is the first in which a diagnosis of calculus of the pancreas has been followed by a successful operation.

I saw the patient with Dr. H. M. Robertson, of Roundhay. She was a lady, aged 57, who had suffered for several months from symptoms which briefly may be enumerated as follows:—

There were steady loss of health, gradual wasting, irregular pigmentation of the skin in patches of the colour of *café-au-lait* (very closely resembling the pigmentation of molluscum fibrosum), persisting attacks of epigastric pain and uneasiness of the type of hepatic colic, though less severe and unattended until very late in the history by jaundice, which was then always trivial though unmistakable, and pain passing through from the front of the abdomen to the middle of the back. There was no rigor or any complaint of sensations of heat or cold. The stools were occasionally "frothy" and "greasy." On examination under chloroform some indefinite swelling could be felt above the umbilicus and a little to both sides of the medium line, though chiefly to the right.

I considered that the evidence warranted a diagnosis of chronic pancreatitis. To explain the onset of this condition I suggested that a pancreatic calculus was present; that its transit down the canal of Wirsung had been attended by the attacks of epigastric colic; and that as it approached the ampulla, an inflammatory condition had been started in the common duct, and a slight jaundice had thereby resulted. Upon this diagnosis I felt justified in basing my suggestion for operative treatment. If chronic pancreatitis were found and no cause was discoverable, the condition could be relieved by drainage of the gall-bladder; if a pancreatic calculus were found, it could be removed either from the pancreatic duct or from the ampulla of Vater, if it had travelled so far. I was bound to admit the difficulties of the case; and though I pressed the question of operation, I felt that I could give no definite undertaking as to its result.

Operation was performed on May 22, 1902. The abdomen was opened two inches to the right of the median line by an incision about seven inches in length, and the fibres of the rectus were split. On opening the abdomen the gall-bladder at once presented; it was tense, well filled, and free from any adhesion; the cystic duct, hepatic ducts, and common duct were all thoroughly examined, the liver being rotated for that purpose. The ducts were all free from adhesion and nothing abnormal was felt. The head of the pancreas was very much enlarged and hard, the body less so, but still larger and denser than the normal. The chronic pancreatitis affected the whole gland, but chiefly the head, and in least degree the tail. The duodenum and the head of the pancreas were carefully examined, and a small lump was then felt, as it seemed, between the two. The swelling did not feel like a stone, and I expressed a suspicion that it might prove to be a growth deep in the ampulla of Vater. I therefore opened the duodenum and exposed the papilla Vateri; this was quite normal in appearance. The lump, however, was felt to be only a short distance beneath the mucous membrane. The papilla was, therefore, laid open, and the cut edges were seized with a small French vulsellum and held apart. At the bottom of the ampulla a small object could be seen, and the knife touching it could be felt to be impinging upon soft stone. A small scoop was therefore passed into the ampulla, pressure was made upon the head of the pancreas close to the duodenum, and the stone was lifted out.

The wound in the duodenum was stitched up with continuous sutures, and the abdomen closed. The patient made a most satisfactory recovery, and is now (July, 1903) quite restored to health, being heavier and in better general condition than she has been for years.

V.—PANCREATITIS.

The subject of inflammation of the pancreas may be said to date its origin from the remarkable paper by Dr. Fitz, of

Boston, published in 1889. It is true that, long before this, isolated observations had been made with reference both to the acute and to the chronic forms of inflammation, but an organised study of the question dates from the publication of the paper mentioned. The most convenient classification of inflammations of the pancreas is the clinical one, which recognises acute, subacute, and chronic varieties.

Acute Pancreatitis.—An acute inflammation of the pancreas may result in diffuse local or general hæmorrhage, in necrosis of the gland or in suppuration. The two former varieties are the most acute; the last merges, at times, insensibly into the subacute form. Acute pancreatitis is recognised clinically as acute peritonitis in the epigastric region. The onset of the symptoms is always abrupt, and their progress rapid. There is a sudden, intolerable, often overwhelming pain in the upper part of the abdomen; collapse, faintness, and vomiting follow in quick succession. In its intensity, and in its character and localisation, the pain can only be compared with that which follows the perforation of a gastric or duodenal ulcer. It is at the first continuous, lasting sometimes without intermission for several hours unless relieved by morphine. In many instances the pain is said to radiate around the lower costal margin of one or both sides to the back. The vomiting is frequently repeated, and occurs very shortly after the onset of the pain. The ejected matter is generally described as "bilious." The temperature is slightly elevated as a rule, though a rise beyond 100° F. or even 101° F. has been occasionally observed. The pulse is almost always rapid and of poor quality. If morphine is given, it may, however, soon regain its tone, and be even slower than the normal. Jaundice is often observed, and is due to an associated catarrh of the common bile-duct.

An examination of the abdomen generally reveals some indefinite epigastric fulness and hardness. The lower part of the abdomen and the flanks are not affected. The patient is as a rule of middle age and stout. A history of gall-stones, or of alcoholic excess, or of indigestion, can frequently be elicited. The urine at times contains albumen or casts. There is rarely intestinal obstruction, flatus being passed without difficulty, and the bowels acting naturally. One sign upon which stress is laid by Halsted is cyanosis of the face and abdominal wall. In one case I have seen this condition; the cyanosis affected the face, abdominal wall, thighs, and buttocks. It will thus be seen that the clinical picture presented by a case of acute pancreatitis shows striking resemblances to certain other acute conditions beginning in the upper part of the abdomen. Among such may be mentioned the perforation of gastric or duodenal ulcers, acute intestinal obstruction high in the small bowel, acute phlegmonous cholecystitis, and acute gangrenous appendicitis. In all of these conditions the previous history of the case is of supreme importance. When any acute catastrophe has occurred within the abdomen, it is only by carefully eliciting the early symptoms that a discrimination between many conditions each closely resembling the others is possible.

I should succinctly depict a characteristic case of acute pancreatitis in the following manner:—The patient is generally very stout, there is commonly a history of gall-stones or indigestion or alcohol. He is suddenly seized with pain in the epigastrium; the pain is agonising and intolerable; collapse follows, and vomiting sets in. The pulse is rapid and thin; the temperature is slightly elevated, but may be irregular. Jaundice occurs frequently. There is no intestinal obstruction; flatus and often fæces passing naturally. At the end of twenty-four hours the epigastrium, which before was tender and rigid, now shows evidence of slight distension, and is more resistant. Cyanosis of the face, abdominal wall, or thighs may be observed. In the most acute cases death occurs in from three to five days; when suppuration, with or without hæmorrhage, occurs, the fatal issue results in from seven to ten days. If the life of the patient be prolonged beyond this, the case may be considered as forming a link between the acute and subacute forms.

Treatment of Acute Pancreatitis.—At the onset a dose of morphine must be administered hypodermically to relieve the excessive pain, but until a diagnosis is made a repetition of the morphine is not desirable. The possibility of relief to this class of case by early operation has not yet been definitely settled by experience, but if only an accurate diagnosis can be made, it would seem only reasonable to suppose that drainage of the pancreatic region from behind would prove of some value. If an exploratory laparotomy is performed for the purpose of diagnosis, one or more areas of fat-necrosis may be found, and the diagnosis may then be made with confidence. Drainage may be effected through an anterior, or better through a posterior, incision. If necrosis of the pancreas is found, as much of the sloughs must be removed as is possible without damage to the healthy portion of the gland remaining. Opie looks on the necrosis as the late result of hæmorrhage. Bœckel in 1900 reported 20 cases of operations upon gangrenous and purulent pancreatitis with 11 recoveries.

Subacute Pancreatitis.—Subacute pancreatitis differs from acute pancreatitis solely in the intensity of its symptoms. The symptoms are the same in kind, though less severe; their onset is less abrupt, their progress less rapid, and their effect on the patient's condition less serious. Pain and vomiting are the earliest symptoms; collapse is generally slight or absent. The epigastric tenderness is well marked, and an ill-defined swelling slowly appears above the umbilicus. Jaundice has been rarely observed. There is no intestinal obstruction, but pus or blood may be found in the motions. The pulse is rapid, 90 to 100; the temperature varies between 100° and 102° F. as a rule; though, when pus is forming, there may be one or more sharp rigors. The general health of the patient suffers greatly. He loses weight rapidly, eats and drinks but little, and is quickly reduced to a condition of extreme feebleness. Albumen is frequently found in the urine.

In the great majority of subacute cases, pus forms either around the pancreas or in the lesser sac of the peritoneum, after the peritoneal covering of the pancreas has been destroyed. Under such circumstances the foramen of Winslow is closed by the inflammatory process, and the lesser sac becomes gradually distended. The pus may burrow in various directions; it may burst into and be evacuated through the stomach or any portion of the bowel; it may rupture into the general peritoneal cavity; or may follow the psoas, causing a psoas abscess.

Treatment.—Subacute pancreatitis must be treated surgically. Drainage should be effected preferably by a posterior incision. The incision begins at the tip of the 12th rib of the left side, and passes obliquely forwards towards the umbilicus. Through such an opening Peters has drained a hydatid cyst of the gland.

An exploratory abdominal operation may first have to be performed in order to confirm the diagnosis. Areas of fat-necrosis should be sought, for their presence is a considerable aid to diagnosis. If a peripancreatic abscess be thus recognised, it will be safer to close the anterior wound and to drain through a posterior incision.

Chronic Pancreatitis.—It is only within recent years that the importance and the frequency of chronic inflammation of the pancreas have come to be recognised. Until Riedel first called attention in 1893 to the association of thickening of the head of the pancreas with cholelithiasis, chronic inflammation of the gland was not recognised as a morbid condition; and even after this time, for several years, the association was considered most infrequent, and of no great surgical importance. In 1900 Mayo Robson, in a lecture published in the *Lancet*, and subsequently in a paper read at the International Medical Congress in Paris, emphasised the surgical importance of chronic pancreatitis, and repeated the statement of Riedel as to the frequency of its dependence upon gall-stone irritation.

Chronic pancreatitis, as seen by the surgeon, is always secondary. It is dependent upon infection extending from the intestinal canal or from the bile-passages, upon the long-standing irritation of pancreatic stones, or upon the invasion of malignant disease, which may be primary in the gland itself, or may extend into the gland from the duodenum or stomach. Certain toxic substances brought by the blood to the gland may set up chronic inflammation; of such are the poisons of syphilis, the organism of tuberculosis, and, it is confidently said, alcohol. Preëminent among all these causes we must, without question, place cholelithiasis. Associated with, and dependent upon, gall-stone disease may be found every grade in the extension of inflammation to the gland. In the slightest cases a little thickening of the head of the gland is found; and that the portion nearest the duodenum is denser and stiffer than the body or tail. In the severer cases the whole head, and a part of the body also, may be enlarged and extremely hard and solid, feeling like a plaster-cast of an enlarged gland. But in all cases the duodenal end is more profoundly affected by the changes which have taken place than is the body. The tail is implicated only in the long-standing and severer instances. Rarely, a chronic inflammation of the pancreas may be the last stage of an acute or a subacute pancreatitis. It is the frequent accompaniment of malignant disease of the gland.

Symptoms and Diagnosis.—The symptoms of chronic pancreatitis vary according to the causation of the disease. In the majority of instances when the inflammation is secondary to repeated attacks of cholangitis the onset is gradual. In each attack some damage is inflicted upon the pancreatic duct, and upon the head of the gland; and long after gall-stones have ceased to pass, or attacks of cholangitis due to cholelithiasis to occur, there may be a repetition of all the symptoms and signs noted in the early attacks. Under these circumstances the cause of the attacks is to be found, not in cholelithiasis, but in the resulting chronic inflammation of the pancreas. In some cases therefore the symptoms and the signs of chronic pancreatitis are identical with, and indistinguishable from, those of cholangitis. In other instances the general appearance of the disease may closely resemble that given by malignant disease of the head of the pancreas. Indeed in the earliest recognised cases, as in one under the care of Mayo Robson in 1892, a diagnosis of malignant disease was made before operation, and apparently confirmed at the operation. It was only after the patient's death that it was recognised that the tumour was due to chronic interstitial inflammation of the pancreas and not to cancer. In other cases, though a resemblance to the symptoms of cholelithiasis exists, a differentation is possible. The pain and tenderness, instead of being over the gall-bladder, are in the mid-line chiefly; pain does not radiate round to the right shoulder, but passes round the costal margin to the left. Jaundice comes on quickly, but deepens much in each attack of pain. As a rule the jaundice in chronic pancreatitis is not absolute; there are variations. In malignant disease jaundice comes on slowly and insidiously, deepens gradually, and is unremitting. In chronic pancreatitis there is a steady loss of flesh and strength, but again the weakness is by no means so swift in onset, nor so rapid in course, as in malignant disease. In one patient observed by me jaundice had existed, with transient lessening and recurring deepening, for over five years. Cancer of the head of the pancreas was excluded simply on account of the duration of the disease.

Courvoisier was the first to point out a fact, which has since been formulated as *Courvoisier's law*, viz., that persisting jaundice associated with a distended gall-bladder is due to cancer of the head of the pancreas. This law has but few exceptions.

The general condition of the patient is almost always poor. The appetite is small and capricious; food even in small quantities causes uneasiness and flatulence. The alterations in the fæces and in the urine, to which reference has already been made, may be observed. Glycosuria is only present in the "interacinar" form of Opie. In a few examples a very slow pulse, varying between 40 and 50 beats to the minute, has been observed.

Treatment.—The only effective and certain treatment of chronic pancreatitis consists in the performance of a cholecystotomy. A prolonged drainage of the gall-bladder and bile-ducts is necessary. It might seem desirable, on being first called upon to deal with this class of case, to perform the operation of cholecystenterostomy, and thereby to drain the gall-bladder into the duodenum or into the colon. As a matter of experience this operation is not attended by the same satisfactory or speedy result as is attained by drainage of the gall-bladder directly to the exterior.

TROPICAL DISEASES: A REVIEW OF RECENT WORK.

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MALARIA.

AN important volume of memoirs dealing with Malaria in India, and based upon work done by Captain James, I.M.S., and Drs. Stephens and Christophers has been issued by the Government of India.[1] The various methods for the examination of the blood and for the study of mosquitoes are first detailed. A good description with illustrations of the Indian species of *anopheles* is given, and the validity of certain species closely resembling one another is questioned, for it is pointed out that small variations of wing-markings, &c., undoubtedly occur, if a number of specimens of a species be examined. It is suggested, therefore, that differences in the eggs and larvæ will ultimately form the true criterion for separating allied species.

Koch, Stephens, and Christophers have shown that in Africa a large proportion of children have malarial parasites in their blood, although the adult population may be free; and it is found that the same holds good for India. Thus, in the tea-planting districts of the Duars between 65 and 75 per cent. of the children under ten years of age have the parasites in their blood, while no infection at all is to be found in the adults.

With regard to the relation between enlarged spleens and malarial infection in India, the following conclusions are arrived at:—

1. A high endemic index in India is always accompanied by a high (usually higher) "spleen rate."

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[1] *Scientific Memoirs by Officers of the Medical and Sanitary Departments of the Government of India.* No. 2. "Malaria in India." By Captain S. P. James, M.B., I.M.S. Calcutta, 1902.

2. A high "spleen rate" may exist in adults *and in children* without a corresponding parasite-infection. These are either (*a*) chronic cases, in which the parasites of the last new infection have gradually died out, but which will be liable to become infected again when opportunity occurs, or (*b*) cases which from repeated infections have become immune to new infection with parasites, but in which the spleen has not yet had time to diminish to its normal size.

3. The "spleen rate" in adults is no indication of the prevalence of malaria. In places which are very malarious it may be very low; in places which are only slightly malarious it may be very high (Calcutta, &c.).

4. Bearing in mind that the spleen is comparatively slow to enlarge and diminish in size, the "spleen rate" is entirely influenced by the "parasite rate." In places where the "parasite rate" decreases at advancing age-periods, the "spleen rate" also does so, although in some places an increase up to a certain age may occur owing to the time taken for individual spleens to diminish to their normal size. In places where the "parasite rate" goes on increasing with advancing age (*i.e.*, less malarious places where no immunity is established), the "spleen rate" also does so, so that while there may be no enlarged spleens among the children there may be many among the adults (Madras, &c.).

The question has been raised whether *malarial cachexia* is really a malarial condition, for the absence of parasites and failure of quinine, &c. would seem to distinguish it from malaria. Rogers[1] points out that Stephens and Christophers have shown that the malarial parasite may be absent for days from the peripheral circulation of acute recent malarial infections in which no quinine has been given; that pigment-granules (melanin) are abundantly present; that the leucocyte-count is the same as in malaria: he is therefore disposed to regard the condition as being really malarial and not due to a secondary infection.

A considerable amount of work has recently been done upon the *leucocytes in malaria*. Rogers[2] has found that while the total number of leucocytes is usually diminished, the large uni-nuclear variety is relatively increased, reaching from 12 to 30 or even 40 per cent., as compared with the normal of 3 to 8 per cent. Daniels[1] remarks that, as regards some points, malaria is more readily studied in England than in the Tropics. The cases being less numerous can be more closely watched, and the effects of the surroundings of the patient and his parasites are not complicated by the possibility of a fresh infection. With both tertian and quartan fevers symptoms occur in accordance with the number of parasites present. The number of parasites necessary to cause symptoms appears to be, for most patients in England, about 200 to 300 per cubic millimetre of blood. In many instances a rest in a warm bed without treatment results in such a diminution of the parasites that no symptoms (*i.e.*, no fever) ensue, and this condition may persist for an indefinite period, the parasites being constantly present in small numbers. In such a patient, however, exposure to cold in some way enables the parasites to multiply sufficiently to cause an attack of fever.

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[1] *Ind. Med. Gaz.*, November, 1902, p. 424.

[2] *Ibid.*, p. 430.

The actual *conjugation* of the micro- and macro-gametes of the malarial parasite has been but seldom witnessed, and Moore's observation[2] of this phenomenon is therefore of interest. While studying the blood of a case of æstivo-autumnal fever, he observed a granular and a non-granular parasite near together in the same field. The pigment of the hyaline body was soon in active movement and four very active flagella were suddenly protruded. One of these soon broke free, and, plunging about in all directions, happened to come in contact with the granular body. After several efforts the flagellum entered and fused with the latter.

Goldhorn[3] states that in protracted cases of tertian fever the *sexual forms* of the parasite differ essentially, and may be distinguished from the ordinary forms of the asexual cycle. They do not segment like the asexual parasites, and are never seen in recent primary infections. Stained with chromatin-dyes, the male gamete is a rather large parasite with a wide achromatic zone in which a mass of unevenly-staining chromatin-filaments lies. The body of the parasite takes basic dyes poorly, and its coarse blackish pigment is brought out more distinctly than in any other form. Before it "flagellates," which it does in a moist chamber, its usually quiescent pigment becomes agitated. The female gamete is still larger when fully developed: it contains a small irregular chromatin-mass situated excentrically, and its pigment is finer than that of the male gamete.

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[1] *Brit. Guiana Med. Annual*, 1902.

[2] *Johns Hopkins Hosp. Bull.*, October, 1902, p. 235.

[3] *Proc. New York Path. Soc.*, Vol. II., No. 5, October, 1902, p. 89.

Moore and Allison[1] have made a comparative study of the *value of methylene blue and quinine* in the treatment of malarial fever. As the cases were admitted to the wards, one was treated with methylene blue and the next was given quinine, no notice being taken of the type of fever, the diagnosis being based upon the microscopical examination of the blood, and the treatment not being begun until the parasites were found. Twenty cases were thus studied, ten being treated with each drug. Pure medicinal methylene blue must be used, not methyl blue, which is stated to give rise to toxic effects. To distinguish between the two a solution of caustic soda may be employed. With methylene blue this gives a deep-violet colour, with methyl blue a purplish-red. Full doses of each drug were administered, 15–40 grains of quinine daily and 20 grains of methylene blue, the latter divided into four doses of five grains each, and with each dose two grains of powdered nutmeg being given to lessen the tendency to strangury.

Of the ten cases treated with methylene blue eight were of the æstivo-autumnal, and two of the double tertian, type. Of the "quinine" cases seven were æstivo-autumnal and three tertian. The average duration of illness in the "methylene-blue" cases was six and a half days after treatment was commenced, and in three this drug failed to cure. The average duration of illness in the "quinine" cases was five and eight-tenths days after treatment was commenced; all were cured, and, in addition, quinine also cured the three cases that failed to react to methylene blue. In three of the cases the methylene blue produced some scalding and pain on micturition. The authors give the following as their conclusions:—

"1. Methylene blue will destroy malarial parasites in many cases, but is less certain that quinine.

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[1] *Med. News*, New York, December 6, 1902, p. 1063.

2. Methylene blue is probably most valuable in chronic cases, but has no advantage over quinine.

3. The effects of methylene blue are ordinarily more unpleasant than quinine.

4. It is useful in cases that cannot take quinine on account of some idiosyncrasy towards it. Its use in cases of pregnancy is undetermined.

5. It is probably valuable in treating hæmaturic and hæmoglobinuric fevers on account of its diuretic action; this has yet to be determined. We have had no chance to test its use in such cases.

6. We believe that quinine is quicker and much more certain, and would rely upon it rather than upon methylene blue."

Kuhn[1] proposes to employ a *serum* used for horse-sickness as a means of cure for malaria. The mode of preparation of the serum is not stated, but for the treatment of malaria from one to a few cubic centimetres of it are injected subcutaneously. As a result of the inoculations Kuhn claims that there occur—(1) Relief of the actually existing paroxysm, the serum being a substitute for quinine; (2) Destruction of all parasites in the blood, and avoidance of relapses; (3) Protection against fresh infection.

The results obtained cannot be said to be very convincing, and the theories of the treatment set forth are very hypothetical. Like others of the German School, Kuhn states that blackwater fever in South-West Africa is essentially due to quinine-poisoning.

Arrhenal, the new cacodylate preparation of arsenic introduced by Gautier for the treatment of malaria, has not been found to be of any service by Manson, who has treated without benefit two cases which at once yielded to quinine. Fraser has also expressed the opinion that the cacodylates are of little value. Cochez[2] of Algiers has likewise failed with arrhenal. He gives details and charts of five cases of malaria treated with this drug in doses of 0·05 to 0·2 centigrams *per diem* without the slightest influence upon the course of the fever—the same cases yielded at once to quinine.

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[1] *Inoculation against Malaria.* By Dr. P. Kuhn. Translated by H. A. Nesbitt, M.A. London: H. K. Lewis. 1902. Price 2s. nett.

[2] *La Presse Médicale*, August 27, 1902, p. 822.

A good review is published by Cardamatis[1] of the hæmoglobinuric fevers of Greece, showing that there are probably several varieties of this condition due to different ætiological causes.

Sir William Macgregor[2] has given an excellent survey of malaria. He is emphatic as to the preventive power of quinine, and says that "no European who cannot, or will not, both as a curative and as a preventive measure, take quinine, should be sent out to Malarial Colonies." With regard to blackwater fever he states that the most successful treatment known to him is the subcutaneous injection of saline solution, 7 grams of sea-salt to 1,000 of water: 100 to 300 c.c. are injected subcutaneously daily and four to six enemata of 200 c.c. may also be given daily.

Under the title of "A Pot of Basil," Shipley[3] draws attention to the properties possessed by the *Ocimum viride*, a labiate plant nearly related to basil. He states that in Northern Nigeria the natives employ it to protect themselves from the attacks of mosquitoes; and that Captain Larymore had informed him that a single plant in a room undoubtedly drove the mosquitoes out, and that by placing three or four of the plants round his bed at night he was able to sleep unmolested without the use of a mosquito-net. With reference to this, Captain James[4] says that "in India the shrubby basil (*Ocimum gratissimum*) is reputed to have the same attributes, and that although it is doubtful whether the popular belief that this tree keeps off mosquitoes and fever has any foundation in fact, the leaves when dried and burnt are certainly a very effective means by which mosquitoes may be killed, and are far superior to the burning of sulphur for this purpose."

KALA-AZAR.

Bentley[5] has recently studied this disease at Nowgong, and has suggested that it is not malarial in nature, but is in all probability a severe form of Malta fever, complicated in a certain proportion of cases with an intercurrent attack of malaria. He points out that—(1) kala-azar during its early stage never exhibits true pyrexial periodicity; (2) during the second stage of the condition quotidian periodicity is common, but the character of the fever is quite atypical of malarial fever, showing a very constant and limited diurnal variation; (3) at all stages the fever is resistant to, and almost unaffected by, quinine, even when given in large doses and over long periods; (4) examination of the blood fails to show the presence of malarial parasites in the majority of instances, while the frequent absence of pigment and of other signs of paludal. infection indicate that that condition is most probably an accidental complication; (5) the morbid anatomy is typical only of chronic pyrexia; (6) investigation shows that the presence of a micrococcus may sometimes be detected in the spleen-tissue; (7) following upon the splenic and hepatic enlargement a chronic low fever ensues accompanied by joint-pains simulating rheumatism with profuse sweating. Moreover, of 25 cases examined by Major Semple by the agglutination-method for Malta fever, 12 gave a complete reaction in dilutions up to 1 in 40, 4 gave partial ones, and only 6 failed to react at all.

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[1] *Le Progrès Médical*, September 13 and October 4, 1902.

[2] *Brit. Med Journ.*, 1902, II., p. 1889.

[3] *Nature*, January 1, 1903, p. 205.

[4] *Brit. Med. Journ.*, 1903, I., p. 677.

[5] *Ind. Med Gaz.*, September, 1902, p. 337.

Rogers,[1] in a criticism on the foregoing paper, suggests that the agglutination-reaction carried out by the sedimentation-method has given fallacious results, and denies that there is any resemblance between the two diseases—kala-azar and Malta fever. Price,[2] also, who is District Medical Officer for Nowgong, and has seen some 2,000 cases of kala-azar, does not agree with Bentley's "Malta fever" hypothesis, but believes the disease to be of malarial origin. He states that he has never seen affection of the joints in the course of the disease.

Basset-Smith[3] also dissents from the view that kala-azar is a manifestation of Malta fever. The chief points of difference between the two are that in kala-azar there is (1) a greater degree of infectivity and more rapid diffusion; (2) a higher mortality; (3) a more constant hypertrophy of the liver and spleen; (4) a greater degree of emaciation and pigmentation produced; (5) an absence of those neuralgias and peripheral neuritic symptoms so important in Mediterranean fever.

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[1] *Ind. Med. Gaz.*, October, 1902, p. 377.

[2] *Ibid.*, p. 379.

[3] *Journ. Trop. Med.*, February 2, 1903, p. 37.

PLAGUE.

The "Report on the Causes and Continuance of Plague in Hong Kong," by Professor Simpson, is interesting and instructive reading. He shows that the whole of Southern China is more or less infected with plague. The proximity of Hong Kong to such an extensively-infected area naturally entails risk of infection, which is increased by the fact that the population of the colony is largely a floating one. Plague, however, is endemic in Hong Kong, apart from any re-infection, this endemicity being mainly dependent upon rat-infection. With regard to rat-infection, enquiries were made of 37 medical men and others resident in various plague-infected districts in China, of whom 30 had found the plague-outbreak to be preceded by a mortality among rats, four were of opinion that the rat-mortality was simultaneous with the plague-outbreaks while the remaining three did not reply, or were uncertain. Thus 90 per cent. of those in a position to judge connect plague-epidemics with rat-infection. The consensus of opinion, that a high rat-mortality is a precursor of plague in man is practically unanimous, not only among medical men in China experienced in plague, but among the Chinese themselves. Dr. Lyall, one of Prof. Simpson's correspondents, states, "It is generally recognised by the Chinese that rats die first. During the year I have often been told that 'men are dying in such and such a street; rats have begun to die in another street, men not yet.'" Dr. Dobson, another correspondent, states that "dead rats are generally numerous just before an outbreak: the Chinese seem to look upon this as preceding plague." In Hong Kong, plague was found to be epizoötic among the rats, and again it was found that rat-plague in a vicinity preceded human plague.

In the infected rats plague-bacilli are present, not only in the blood and tissues, but also in the intestinal contents, urine and mucus of the mouth in a large proportion. This may give the clue to the vexed question as to how human beings become infected from the rats. The rats soil food, clothing, and other articles with their excreta containing the plague-bacilli, and, by ingestion or otherwise, human beings coming in contact with the infected materials run a great risk of contracting the disease. A number of experiments were carried out with rats, calves, pigs, monkeys, sheep, hens, ducks, geese, turkeys, and pigeons, showing that all these animals and birds may be infected by feeding upon plague-material: it seemed to make no difference what the source of the plague material was. Thus plague-material from man will give plague to pigs, calves, hens, ducks, geese, rats, sheep. Plague-material from hens will give plague to the other birds named, and to rats and pigs; that from the rat to monkeys and hens, and so on. A fact to be noted is that, after the infection enters the body by the alimentary canal, a considerable period sometimes elapses before the animals show any marked signs of illness; in the case of the pig this interval may be a month, or even more. Prof. Simpson remarks, that "plague is consequently not a disease confined to man. The infected material from him is liable, if eaten by rats or other animals, to give them plague; and they in their turn are likely to spread it among the animals and poultry of the farm-yard or house-yard in which these are kept, and again pass it on to man. And the rat is apparently the most important agent in disseminating plague; it has relations both with man's house and with the premises on which the animals mentioned are kept." It is to be noted that in the endemic centres of plague, in Persia, India, and China, the people have a very intimate association with their domestic animals and poultry.

Calvert[1] has made a systematic examination of the blood for bacilli during the course of an attack of plague. The plan adopted was to take blood from the ear, at intervals of four hours, until the death or recovery of the patient, and to examine it in smears and by cultures. Smears were stained with Gram's gentian violet, examined, then decolourised and lightly re-stained with methylene blue or carbol-fuchsin. The object of this was to bring out the bipolar staining; without some such preparation as absolute alcohol or Gram's method the bacilli often fail to take a bipolar stain. In all, 36 cases were examined, of which 32 were fatal and 4 recovered. With regard to the results obtained, the *Bacillus pestis* was found in all the fatal cases, in smears and cultures, within 24 hours before death; in 7 cases within 48 hours, in 5 cases within 72 hours, in two cases within 96 hours, and in 1 case within 120 hours. Cultures seemed to be of little more value than smears, cultures and smears being as a rule positive on the same day. In fatal cases bacilli are practically always found in the peripheral circulation at some time during the course of the disease. In one of the cases that recovered bacilli were found to be present in the blood during 45 days. Bacilli were found in the blood in all the cases that recovered, so that their presence is not of much value as regards prognosis. The development of the septicæmia can be watched by this method; at first only a single or a few organisms can be found in a smear; four hours later a distinct increase in the number of bacilli can be noted. This increase continues until just before death, when the blood may be in some cases almost a liquid mass of bacilli.

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[1] *Centralbl. f. Bakt.*, I. Abt. Origin, Bd. XXXIII., 1903, p. 247.

DENGUE.

Harris[1] describes the clinical types of dengue occurring in Madras as follows:—

"1. Mild aching pains in the limbs and in the loins, rise of temperature to about 103°, closely resembling an ordinary fever resulting from exposure, and yielding to mild diaphoretics with a stiff dose of sodium salicylate. No eruption.

"2. Excruciating pain in the joints of both upper and lower limbs of *one side* only with or without eruption. Temperature rises to 105°, lasts, fluctuating, three to five days and yields. Hot fomentation relieves pain. I suffered from this form.

"3. All the joints are affected. Pain unbearable; patient helpless, screwed down to bed. Temperature very high; sometimes becomes hyperpyretic; death may follow if temperature be not promptly brought down; generally without eruption.

"4. Pain much less marked. Temperature very high, or moderately so. On the second, third, even on the fourth day eruption occurs, in character resembling roseola, or erythema, and lasts for two to many days."

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[1] *Indian Med. Rec.*, December 31, 1902, p. 769.

There has recently been a considerable epidemic of this disease in Burma. From observations made in this epidemic Pridmore[1] believes the incubation-period to average about three days. The secondary rash invariably appeared on the fifth or sixth day, and, in at least 75 per cent. of the cases, enlargement and tenderness of the cervical, axillary, inguinal and supracondylar lymphatic glands preceded or accompanied the secondary rash. Conveyance of infection through the mosquito, as suggested by Graham, could not be confirmed, nor was Graham's parasite in the blood detected.

MYCETOMA.

Lieut.-Colonel Maitland[2] points out that it is not uncommon to find the femoral lymphatic glands enlarged in cases of mycetoma of the foot, but this enlargement is as a rule merely the result of irritation, and the changes found in the glands are due to hyperplasia of the connective-tissue elements. Occasionally, however, a true infection of the glands takes place, and two or three instances of this are quoted. When once the glands have become infected, the disease spreads to the surrounding tissues, and a condition of things is produced exactly resembling that which is seen in the original focus of the disease in the hand or foot, and almost hopeless from the point of view of treatment. Daniels[3] states that he has met with cases of mycetoma in British Guiana.

BILHARZIA DISEASE, ANKYLOSTOMIASIS, &C.

Loos[4] believes that the mode of infection with the bilharzia is by the skin. He has not yet actually demonstrated this, but against the view that infection is by the mouth he adduces the fact that the embryos are killed by even a momentary contact with a weak acid. Madden[5] describes cases of rectal papillomata containing the bilharzia, which was evidently the cause of the growths. Manson[1] records a case of bilharzia from the West Indies, the ova having lateral spines, while Williamson[2] has met with a case in Cyprus, the ova in this instance having the usual terminal spines.

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[1] *Ind. Med. Gaz.*, October, 1902, p. 380.

[2] *Ibid.*, November, 1902, p. 417.

[3] *British Guiana Med. Ann.*, 1902.

[4] Egyptian Medical Congress, December, 1902.

[5] *Journ. Trop. Med.*, January 1, 1903, p. 1.

A remarkable outbreak of *ankylostomiasis* in England has been recorded by Boycott and Haldane.[3] It occurred in the Dalcoath Mine in Cornwall, and affected a number of men. The Cornish miners visit all parts of the world, usually, however, returning home after an absence of a few years; and it is surmised that some of those who had travelled abroad contracted the disease, and so brought the infection home.

Ozzard[4] remarks upon the serious nature of ankylostomiasis and the necessity for adopting stringent measures to stamp out the disease. According to him the best treatment is the administration of thymol in repeated doses at stated intervals, either in the form of a few large hourly doses, or, better still, of a prolonged course in doses of 20 to 30 grains daily, interrupted at times with a course of alkalies and bitter tonics. Areca-nut has proved a failure in his hands. Ozzard has been able to confirm Giles's observations regarding the production of sexually-mature worms outside the body.

Filariasis.—Low has established the fact that the embryo forms of Filaria Demarquayi and F. Ozzardi are absolutely identical, but Ozzard does not consider this sufficient to establish their identity as species. Ozzard points out that the distribution of these two parasites is entirely different, and he describes a parental form of F. Demarquayi which seems to be quite distinct from the so-called F. Ozzardi.[5]

Trypanosomiasis.—Manson records another case of human trypanosomiasis. The patient, a lady who had resided on the Congo, was seen by him in July 1901, and suffered from fever and transient erythema; the spleen and liver were not noticeably enlarged. She was seen again in April 1902, and had much the same symptoms. Recently, Dr. Sims, of

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[1] *Brit. Med. Journ.*, 1902, II., 1894.

[2] *Ibid.*, p. 956.

[3] *Journ. of Hygiene*, III., No. 1, p. 95.

[4] *Brit. Guiana Med. Ann.*, 1902.

[5] *Ibid.*

Leopoldville, has found trypanosomata in the blood. Trypanosomiasis would seem to be common in this part of Africa, for at least four members of the small European colony there have been found to be affected with it. In two of the cases there is a history of a bite of an insect, followed by fever, and Manson suggests that this may be the poisonous tick described by Livingstone as occurring at Tete and other places on the Zambezi.[1]

SLEEPING SICKNESS.

The joint commission upon this disease, sent out by the Royal Society and the London School of Tropical Medicine, consisting of Drs. Low, Castellani, and Christy, has now finished its labours. Low has practically proved that the Filaria perstans is not the cause of the disease, while Castellani has isolated a specific streptococcus in a number of cases.[2] There seems every reason to suppose that this is the same organism which was previously isolated by the Portuguese Commission (1902), and termed by them the "hypnococcus." More recently Castellani[3] has discovered in the cerebro-spinal fluid of sufferers from sleeping sickness (70 per cent.) a peculiar form of trypanosoma, and this was once found also in the blood. The discovery of this organism is confirmed by Bruce,[4] who found the trypanosoma in every one of 38 cases examined (cerebro-spinal fluid), and in the blood in 12 out of 13. Castellani considers this organism to be different from the one causing trypanosomiasis (*see* above). The method of demonstrating the organism is to draw off a considerable quantity (15 cc.) of cerebro-spinal fluid and treat this with the centrifuge for ten minutes. The deposit is then examined with a low power of the microscope, and trypanosomes are easily seen, if present, as they exhibit active movements.

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[1] *Brit. Med. Journ.*, 1903, I.

[2] *Journ. Trop. Med.*, March 16, 1903, p. 85.

[3] *Brit. Med. Journ.*, 1903, I., p. 1431.

[4] *Royal Society*, May 14, 1903.

CASES WITH COMMENTS.

I.

A CASE OF THREATENED PUERPERAL ECLAMPSIA.

By EDWARD ELLIS, L.R.C.P.E., L.R.C.S.E.

In the February number of THE PRACTITIONER there appeared a most interesting article by Dr. Fothergill on "The Ætiology and Treatment of Puerperal Eclampsia," giving the theories of several eminent medical men as to its causation and treatment. From amongst them I single out the theory of Dr. H. O. Nicholson, who believes that the eclamptic state is due to thyroid inadequacy causing an auto-intoxication from inefficient metabolism, and imperfect excretion. The resulting toxæmia leads to injury of the kidneys, causing albuminuria and more or less suppression of urine.

The following notes of a case which came under my care in March of this year, when I carried out the treatment suggested by Dr. Nicholson, will prove of interest :—

On March 3rd, a patient, Mrs H., called to ask me to attend her in her confinement. She was 27 years of age, and a primipara. She was seven months pregnant. I noticed that her face and hands were swollen, and on making further examination I found extensive œdema of the legs also. Very little urine was being secreted, not more than 15 to 20 ounces in the 24 hours. There was a good deal of headache, vomiting, insomnia, and failing eyesight, the patient being unable to read or to do needlework. I examined the urine the next day (March 4th) with the following result :—on boiling, it was nearly solid, of the consistency of thick cream. The specific gravity was 1030. It contained 21 per cent. of albumen as measured by Esbach's albuminometer. The patient was ordered to bed, put on a purely milk diet with barley-water and plenty of plain water. A free purge was given and a hot bath every other night. A thyroid tabloid (5 grs.) was ordered to be taken three times a day.

The following notes show the condition of the urine :—

	Sp. gr.	Albumen.	
Mar. 5th.	1020.	12 per cent.	Marked increase in the urine (two pints) and free action of the skin.
„ 10th.	1020.	5 „	
„ 16th.	1030.	20 „	The increase of albumen I think was due to feeling a chill after the hot bath.
„ 19th.	1018.	4 „	Patient allowed milk-puddings, chicken-broth, tea, coffee, and fruit.
„ 26th.	1010.	3 „	Patient allowed fish, chicken, pigeon, bread and butter.
„ 28th.	1010.	1 „	Patient downstairs for meals and resting on the couch, and taking carriage exercise.
Apr. 2nd.	1010.	½ „	
„ 12th.	1030.	½ „	

On April 16th, labour commenced in the forenoon, a foot presenting; and at 4 p.m. she was delivered of a still-born female child, which must have been dead some weeks. The patient made an uninterrupted recovery, and on testing the urine on April 24th the specific gravity was 1010, and it was free of albumen.

The thyroid was given three times a day up to the 19th March, and then reduced to 10 grs. a day. Soon after the treatment was adopted the œdema entirely disappeared, the eyesight became normal, and the headache, sickness, and insomnia passed away. From the first day of treatment the urine became normal in quantity, and there was free diuresis. The thyroid caused no palpitation or shortness of breath. I examined the urine after the patient had been up and about and found the specific gravity 1018 and no albumen.

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II.

A CASE OF DOUBLE HARE-LIP WITH ABSENCE OF NASAL SEPTUM.

BY S. DAVIDSON, M.D.

[With Plate I.]

The accompanying photograph is that of an infant born on 28th May last. It presented the following abnormalities at birth. There was a complete double hare-lip, and also a cleft in the premaxilla, but no cleft in the palate. The prolabium was entirely absent, so that a complete gap existed below the nose, the upper lip stopping short on either side at a point vertically below the outer side of the ala of the nose. In addition, the nasal septum was entirely absent. There seemed to be no cartilaginous tissue in the nose, which was perfectly soft and flat, and showed no prominence in profile.

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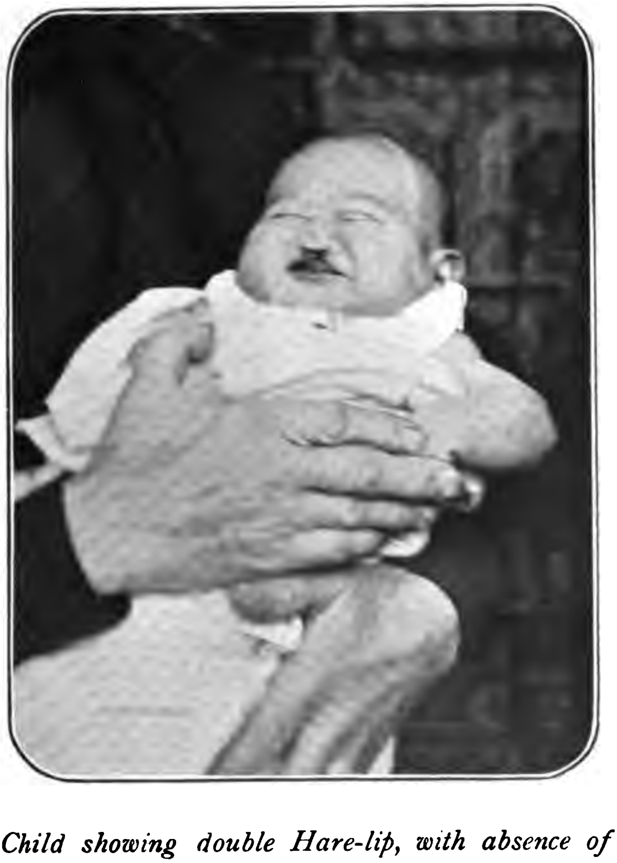
PLATE I.



Child showing double Hare-lip, with absence of Nasal Septum.

Public Health.

ENTERIC FEVER AND INFECTED BLANKETS.

BY LOUIS C. PARKES, M.D., D.P.H.,
Medical Officer of Health, Chelsea.

TOWARDS the end of May, 1903, Dr. Collingridge, Medical Officer of Health of the City of London, reported that he had reason to believe that an outbreak of enteric fever on the training ship "Cornwall," stationed in the Thames, was due to the use of blankets from South Africa, which had been sold out of the army, and subsequently supplied by an East-End firm of merchants to the authorities of the "Cornwall." The cases occurring on the "Cornwall" were some ten in number, and occurred early in April. An investigation of the cause of the outbreak failed to establish any connection between the illness and the more usually occurring sources of infection, such as water, milk, food, &c., but suspicion was directed to certain blankets which had been recently taken into use, and which, as before said, had been bought from agents who had received them from army sources. Some of these blankets were soiled and stained, and were submitted to Professor Klein, F.R.S., for bacterioscopic examination, with the result that abundant *Bacilli typhosi* in an active condition were reported to be present.

Dr. Collingridge ascertained that the blankets sent to the "Cornwall" formed only a small part of a large consignment of army blankets which had been received in London from South Africa, and he at once traced to their ultimate destinations all over the country the remaining bundles of blankets which had been sent out by the City firm who were the original purchasers; and he communicated with the Medical Officers of Health of the various districts to which he had reason to believe these blankets had been sent. In a later report to the Corporation of the City, Dr. Collingridge stated that he had ascertained that about 20,000 of these army blankets had been sold to 289 customers in all parts of the kingdom. Large numbers had been burned, others had been disinfected by the local sanitary authorities, whilst in several instances the blankets had been returned to the wholesale dealers in the City. All the blankets remaining in stock and those returned to the wholesale agents were in course of disinfection, and would be subsequently washed before they would be released. Bacterioscopic examination of blankets sent to the provinces, as well as of some in London, had shown them to contain typhoid-bacilli in large numbers. An outbreak of enteric fever had been traced directly to their use in a Midland town. There would probably have been other outbreaks had it not been for the fact that most of the purchasers had held the blankets in reserve and not brought them into actual use before their infected condition became known.

The official explanation of how these army blankets, which had been used during the South African campaign, came to be offered for sale to the general public, was supplied by the Secretary of State for War in the House of Commons. Mr. Brodrick said that the General Officer Commanding in South Africa reported that in October, 1902, owing to the rapid demobilisation of the troops, an enormous stock of unwashed general service blankets, returned to store by the troops, was deposited at the Capetown Ordnance Stores, stacked in the open under tarpaulins. Efforts were made to have them washed and properly stored; but, meanwhile, the stacks took fire by spontaneous combustion, and endangered the entire Ordnance Depôt. Prompt action became imperative, and the chief ordnance officer gave instructions for large numbers of those in the worst condition to be destroyed by fire, and accepted tenders for the sale of 80,000, provided they were removed within three days. These blankets were stated not to be unserviceable, although they had not been washed. The amount realised by the sale was 1,495*l.*, or about 4½*d.* apiece.

From the facts above related, it will be noticed that a period of at least six months must have intervened between the last use of these blankets under service conditions (some time prior to October 1902) and the outbreak of enteric fever on the "Cornwall," early in April, 1903. It does not seem even yet to be definitely known if these blankets, or any of them, stored at Capetown in October, 1902, were those which had been in use in the field hospitals, or whether they were those which were supplied to the forces in the field. In the former case, it appears that hospital blankets are by regulation required to be "stoved" before they are sent to store. The "stoving" process probably means nothing more than the application of a certain amount of dry heat to the articles in the field hospital stoves—a process known to be highly unreliable, if the destruction of pathogenic bacilli is aimed at, and not likely to be very thoroughly applied under the conditions of field service. It is quite easy to understand then how even "stoved" hospital blankets might prove infective, and be found to contain large numbers of *Bacilli typhosi.*

The infective condition of the blankets used by troops on field service—men presumably in good health—is no doubt explained by the undoubted prevalence of mild or "ambulatory" enteric amongst the soldiers during the recent campaign. A large number of blankets must have been infected by these mild cases where the men continued on active duty all the time, and also by others who, having contracted the disease in a more severe form, were passing infective excreta before they could be drafted to the field hospitals. It seems also probable that, under the conditions of close storage under unfavourable conditions at Capetown, where the heat engendered by the fermentation of the organic matter in the closely packed blankets was sufficient to set up spontaneous combustion, infected blankets may have been the means of infecting others with which they were packed, which had so far escaped specific fouling.

On consideration, then, the fact of so many of the disused army blankets being found to be teeming with enteric bacilli is easily intelligible, having regard to the sanitary conditions which prevailed throughout the South African campaign, and the general prevalence of enteric fever amongst all classes of troops. It is more surprising to find that the enteric bacilli in the infected blankets should retain their activity for such a lengthened period as six months or more. Perhaps the best known case of persistent infection in clothing is that given by the late Sir R. Thorne-Thorne, and mentioned in the Report of a Committee of the Clinical Society of London on

Periods of Infection and Contagiousness of certain Infectious Diseases (Suppt. to Vol. XXV., 1892). In this case the clothing of a woman who died of enteric fever at Toronto, Canada, in September, 1873, was brought home by her husband to an Oxfordshire village, on November 20, 1873, the woman's clothing not having been previously cleansed or disinfected. The village was free from any enteric fever at that time, but on the 29th and 30th November the brother and mother of the man, who resided in the house, were attacked with typical enteric, and on the 5th December a female relation, who had taken to her own home some of the dirty things to wash, was also attacked. In this case, then, probably some three months elapsed between the period of soiling of the clothing and the infection of the subsequent cases. In the interval, it is probable that the clothing was for the most part packed into a box or trunk under conditions which excluded the action of light and air upon the specific organism.

The inquiry instituted by Majors R. H. Firth and W. H. Horrocks, R.A.M.C., into the influence of soil, fabrics, and flies in the dissemination of enteric infection, which was conducted at the Hygiene Laboratory, Army Medical School, Netley, in 1901 and 1902, throws considerable light on the persistence of infection in specifically-soiled clothing. These experiments covered a long period of time; they were conducted with extraordinary care, and with all the attention to minutiæ in detail which the experience of the gifted authors could bring to bear upon them; and the identification of the *Bacillus typhosus* was in every instance established by cultural and agglutination tests of the most modern application, which were either unknown to or unpractised by some of the former workers in this subject—a fact which accounts for some of the discrepant results hitherto recorded, it being not always possible to claim with certainty that the enteric bacilli said to have been isolated were not modified strains of *Bacillus coli.*

Three of Majors Firth's and Horrocks' experiments with fabrics were carried out by infecting (1) khaki drill cut from a soldier's coat, (2) khaki serge, and (3) ordinary blue serge, with a rich emulsion of a stock *Bacillus typhosus,* the emulsion being poured over each fabric. The fabric was then placed in a glass dish, and left on the verandah exposed to light and air.

There it rapidly dried. In two other experiments a piece of khaki drill was fouled (4) by rubbing into it 5 c.cm. of a freshly-passed liquid enteric stool to which had been added an emulsion made from a 24-hours' growth of *Bacillus typhosus* recently obtained from a similar stool; and another piece of khaki drill was fouled (5) by rubbing in the solid material from a recently-passed enteric stool. As in experiments 1, 2, and 3, the fabrics were placed in glass dishes and left on the verandah to dry, exposed to light and air.

At varying times a piece of the fabric in each of these five experiments was cut off with sterile scissors, and placed in plain sterile broth. After incubation at 37° C. for 24 hours, cultures resulted which frequently not only showed gas bubbles on the surface, but were distinctly offensive. Loopfuls of the culture were then stroked in concentric rings on to alkaline glucose litmus-agar plates, 3 to 4 plates being inoculated from each loopful of the broth, and 6 to 15 plates being stroked from each culture. The plates were then incubated with covers downwards at 20° C. for four days, and the resulting colonies examined. Suggestive colonies were then carefully fished, and made into an even emulsion with a loopful of sterile broth on a clean cover-glass, and examined as a hanging drop to ascertain that no false clumps were present. The cover-glass was then removed, and with the same needle one loopful of enteric serum, obtained from Berne, diluted to 1 in 500, was added to the emulsion, this procedure converting the serum into a dilution of 1 in 1,000. If instantaneous agglutination occurred, the colony was planted out on an agar slope, and the resulting growth subcultured in the various media (some 12 in number) essential for the diagnosis of *Bacillus typhosus*.

The results of these experiments, as regards the recovery of the enteric bacillus, are as follows:—

Experiment 1.—The bacillus was recovered up to and on the 74th day after inoculation, but not later.

Experiment 2.—Recovered up to and on the 87th day, but not later.

Experiment 3.—Recovered up to and on the 78th day, but not later.

These three experiments gave nearly identical results.

Experiment 4.—The bacillus was isolated on the 12th and 17th days, but not later; not isolated on the 2nd, 6th, and 9th days.

Experiment 5.—Bacillus isolated on the 2nd and 9th days, but not later.

It is evident, then, that in fabrics fouled with enteric stools, the enteric bacillus possesses much less vitality than it does in fabrics inoculated with a stock emulsion. *Bacillus coli*, *Proteus vulgaris*, and other bacteria of putrefaction no doubt arrest the life and growth of the enteric bacillus. In these experiments the fabrics were exposed to light and air, and became very dry. They are instructive as showing that enteric bacilli, when unmixed with other bowel-organisms, may retain their vitality for long periods under most unpromising conditions, but they do not throw much light on the possibility of persistence of infection in blankets fouled with enteric excreta and kept away from light and air, conditions under which the army blankets at Capetown were known to be stored.

Further experiments might be undertaken to elucidate the question of the possibility of the survival of *Bacillus typhosus* in an actively-virulent form in clothing fouled with ordinary enteric excretions under such conditions as must have prevailed in the Capetown Army Depôt. In the meantime the obvious lesson to be derived from recent experiences is that it is unsafe and unfair to the public for the military authorities to sell out after a campaign any blankets, bedding, clothing, sheets or tents which have been used in the field, whether in ordinary field service or in field hospitals, which have not been disinfected by steam or boiled in water to the satisfaction of some responsible medical officer. If the disinfecting appliances available are not such as are recognised as adequate for thoroughly efficient disinfection, then all such stores should be destroyed by fire as soon as they cease to be of use. From the point of view of public health immediate destruction is far preferable to transmitting such goods long distances to the base, and their storage there under conditions likely to propagate and intensify any infection they possess.

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Notes by the Way.

Treatment of Gout.

We were compelled by want of space in our July number to hold over our comment on the treatment of gout to the present issue of THE PRACTITIONER. The exact treatment adopted by the physician will depend to a considerable degree on what theory of the disease he has formed, unless indeed he rely upon a pure empiricism. If he holds that it is due to an accumulation of uric acid in the tissues, he will use some remedy directed to washing this substance out of them. In oblivion of the fact that it is not pure uric acid, but urate of sodium, which forms the actual deposits, a number of drugs which act as solvents of the free acid in a test-tube have been lauded as remedies for gout. Of such we may instance piperazine, lycetol and similar substances. We do not ourselves believe that these substances produce any effect whatever on uratic deposits. Alkalies are administered with the same object of producing soluble salts of uric acid. It seems permissible to doubt whether, when once a deposit has formed, it does any further harm, and whether therefore much attention need be directed to removing it. Whether the drugs just mentioned prevent to any extent the further deposit of the peccant matter is also somewhat doubtful.

If we believe that it is consumption of poisonous materials in our food that causes the disease, then we must proceed to cut off those articles of diet which contain most nuclear products. On this point we have perhaps the most direct contradiction of opinion to be found among the contributors to our Special number. Dr. Haig holds that meat and tea are the direct causes of gout; if we cut off these articles of diet, the disease necessarily ceases. Dr. Luff, on the other hand, denies the morbific action of these common articles of food, championing their cause and attacking their traducers with some approach to acrimony.

If on the other hand it is decided that the cause of the malady is some morbid decomposition taking place in the alimentary canal, it is reasonable to administer some of the drugs which are credited with acting as intestinal antiseptics. But of one thing there is no doubt, and that is that we should not lose sight of the principle insisted on in the sentence from Bacon's Essays so appositely quoted by Sir Dyce Duckworth, that we must treat the patient and not the disease. This is a universal maxim, but in no case is it more important than in gout. The defective nature of our theoretical knowledge of this disorder is emphasised by the failures that result from treating it on general principles. What suits one patient disagrees with another. Literally, what is one man's meat is another man's poison. We cannot with any pretence of accuracy say that this food is gouty and that is not. Treatment in any individual case is largely a matter of experiment.

Far more important than the scrupulous observance by the patient of rigid dietary regulations is careful attention to the ordinary rules of health; he must not debilitate himself by practical starvation, nor exhaust himself by over-exertion. Excess in food and in any other indulgence must be avoided, but particular kinds of food should only be forbidden when they are proved to be injurious. No hard and fast dietary can be laid down in advance. After all, or before all, the patient has to live, and to do so he requires, like other animals, a due supply of the essential food-materials — proteids, fats, carbohydrates and salts.

With regard to these last, there can be little doubt that many persons do themselves much injury by the quantities of medicinal waters of all kinds which they persist in swallowing, as aperients and otherwise. These much-advertised beverages are best avoided except when ordered by the medical adviser. And the same may be said of many of the other purgative medicines constantly taken. We feel confident that they are often taken unnecessarily, and that in such instances they are actually harmful. The form of constipation that results from abuse of purgative drugs is only too well known to medical men.

What is to be said of our purely empirical treatment, of which the use of colchicum is the principal feature? The action of this drug is not well understood. Dr. Haig states that it increases the alkalinity of the blood, but he apparently considers it harmful rather than otherwise. It is also said to act as a diuretic, and may in this way tend to eliminate some of the poisonous material that causes the disease. Experience certainly seems to show that it is of use in the acute stages of gout. As to its prolonged use, Dr. Toogood relates that he has not seen any ill effects in cases in which colchicum was administered inadvertently for long periods of time. The effects of its administration, however, need careful watching, as too large doses may certainly produce undesirable depression.

With regard to the use of salicylates opinions also differ. Dr. Haig is an upholder of these remedies : Dr. Luff is not enthusiastic for them. We think that in some cases they appear to do good, but that in gout they do not play anything approaching to the same important part which they do in rheumatism. The employment of salicylate of potassium rather than of sodium seems suggested in view of the contra-indication to sodium-salts existing in gout. Aspirin appears to be a form of salicylate which deserves a trial in all conditions in which these salts are indicated; it seems to have a good effect in relieving pain in some chronic joint-troubles as well as in the acute attacks.

Treatment of gout at various spas is now very frequently recommended. That it does good in suitable cases is unquestionable, though it is impossible to determine exactly how much of the improvement results from the use of the waters to drink or bathe in, and how much to the regular course of life followed during the "cure." In our Special number Dr. Bannatyne gave a detailed account of the treatment as carried out at Bath, and we include in our present issue the first part of a paper by Dr. London of Carlsbad dealing very fully with the nature of gout. The concluding portion of this article describing the advantages of this well-known health-resort among others, and discussing the treatment of the disease in general, will appear in September. Dr. London has appended to his paper a very full bibliography on the subject of gout, which should be of great use to all who wish to consult the literature of the subject. It is important to remember that great care is necessary in the selection of suitable cases for hydropathic treatment. Acute cases should not be sent away during these attacks, and much harm may be done to weakly and debilitated patients by unwise resort to baths and waters. We cannot too strongly dissuade sufferers from rushing to fashionable spas on their own initiative without reliable medical advice.

It is often a question in the presence of an acute attack of gout, with its agonising pain and mental irritability, whether we are justified in administering morphia. Dr. Luff advises that this should only be given as a last resource, after trial of hot applications and other less formidable remedies. That there is some danger in the use of morphine in view of possible renal lesions is undoubted, but we venture to think that it is exaggerated. Many writers have even advised the employment of morphine in attacks of uræmia itself. Nothing seems capable of affording equally speedy and certain relief to the acute pain of gout, and of the imperative duty of relieving pain of this intensity there is no question. Morphine should be a last resource, but we should not unduly delay in having recourse to it. The danger of creating a craving for the drug by giving a single injection for a definite end is inconsiderable.

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The Cause of Cancer.

OUR discussion of the cause of cancer in the May number of THE PRACTITIONER has brought us in a little correspondence on the subject which is not without interest. In the first place we have to thank Dr. Braithwaite for a copy of his paper on the production of the disease, in which he maintains that there are several factors, all or several of which must be present in order that cancer may originate. The most important is excess of salt in the diet. This substance is a stimulant to the tissues and tends to accelerate their growth. If much salt be constantly taken, then, given certain other predisposing causes, cancer will result. The other conditions necessary are:—Firstly, a state of over-nutrition resulting from excess of food, especially animal matter; secondly, the presence of effete material in the cells, such as occurs in old age; and thirdly, the action of some irritant, such as injury of any kind. Dr. Braithwaite considers that this theory coördinates all the known facts in the causation of the malady. We will not criticise the theory further than to point out that the action of even one cause is difficult enough to follow out in a living body, and that to trace the interaction of four causes, as here suggested, would be well-nigh hopeless. Hence as a working hypothesis the theory has evident drawbacks. We cannot, however, refrain from setting alongside of this theory the exactly-contrary views held by another worker in the field of cancer. The *Medical News* (April 25, 1903) contains the following note:—"Captain Rost, of the Military Medical Service, who has been investigating malignant cancers (*sic*) bacteriologically at the Rangoon Hospital for three years, announces what is believed to be an important discovery. He has found in both carcinomata and sarcomata distinct germs of saccharomycetes, which can only develop when the natural chlorine in the tissues falls below the normal quantity. Following this clue, Captain Rost devised a treatment to reënforce the chlorine by special diet, enabling large quantities of common salt, which contains chlorine, to be absorbed. He has experimented with eight patients. One was completely cured, and the condition of the others was improved." The Editor of the *Monthly Cyclopedia of Practical Medicine*, from which we have taken the above quotation, states that he has been unable to trace the exact source from which it is derived. Its chief interest lies in its direct antagonism to Dr. Braithwaite's view, and we confess that we merely quote it as an *argumentum ad hominem.*

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Varieties of Tumours and the Parasitic Theory.

DR. A. T. BRAND also sends us a copy of a paper in which he calls our attention to his view that the argument against a parasitic causation of tumours drawn from the multitude of different growths is "the merest quibble." It may be so, but it still appears to us to constitute a difficulty. If there is one and the same parasite at work in all carcinomata, how is it that the secondary growths in each variety so exactly (as a rule) imitate the characters of their parent tumours? It seems necessary to suppose that actual fragments of the growth are carried away and develop on their own account; and thus we are at once brought up against a method of spread of infection to which we have no known analogy among recognised infective processes. With regard to Dr. Brand's further insistance on the distinction between malignant and benign tumours, we are not at all sure that pathologically any hard and fast line can be drawn. Clinically, it is easy enough, but on pathological grounds alone it is very difficult to say exactly where, for example, a fibroma ends and a sarcoma begins. To say that a fibroma has "become malignant," or to call a chondroma which has formed metastases a "chondrosarcoma," does not help in understanding the nature of the disease, however comforting the words may be clinically.

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Optologists.

THE English language is evidently in a flourishing condition if rapid growth is to be taken as a test. We fear, however, that some of the recent formations can only be described as morbid. Such terms as "Uricacidæmia" and "Appendectomy" are certainly unhealthy. The newest gem of diction is "Optologist," a term which it is sought to apply to prescribing opticians. As our readers will no doubt recollect from the days when they pursued their studies in the classical languages, the Greek word ὀπτὸς means "baked" or "roasted," and an optologist should be a specialist in the science of baking. Can it be that a subtle sarcasm lurks in the newly-coined term, and that it gently indicates that those who apply to opticians instead of to qualified ophthalmic surgeons to have their sight corrected are to be considered "half-baked," or that they are likely, as the result of their confidence, to be "done brown"?

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Drugs and the Hospital Sunday Fund Administrators.

WE hear with regret that the authorities who administer the Hospital Sunday Fund are endeavouring to cut down the expenses of some of the London Hospitals by insisting on a reduction in the amount spent on drugs. We cannot think that this course is wise or proper. A committee of laymen cannot know anything about the use of drugs, and are therefore in this case interfering in matters beyond their province. By seeking to restrict hospital officers in the use of remedies they run the risk of either causing the hospitals to use inferior drugs on account of their cheapness, or of preventing the physicians from making use of what they consider needful for the treatment of disease. In either case the effect of the interference will be pernicious. We hear of one hospital recommending its officers to limit their prescriptions of such a useful remedy as extract of malt to a definite small quantity in each case, not because larger doses would not be more beneficial to the patients, but solely because the drug-bill must be kept down. Similarly, expensive remedies will be discouraged, not because they may not be the best, but because they are costly. We do not think that it is in this way that subscribers to hospitals would wish the needful economies to be effected. They would prefer that the money should be spent in doing the greatest possible good to the patients, rather than that it should be economised on any system of red-tape devised to suit a central board.

Practical Notes.

TREATMENT OF TRIGEMINAL NEURALGIA.—Many cases of neuralgia of the face are dependent upon local troubles in the teeth, in the frontal and other sinuses, or arising from a dacryocystitis. In such cases removal of the cause is needed to cure the neuralgia. Other instances are produced by syphilis or malaria, or are associated with tabes dorsalis: in the two former specific treatment is to be adopted by means of mercury and iodides, and quinine respectively, while in tabes also antisyphilitic remedies are sometimes successful. Apart from these direct causes, there are a number of mild cases which will yield to warm applications, or to lotions of aconitine or opium. If medical teatment fail, then recourse must be had to nerve-stretching or neurectomy. Before, however, we condemn a patient to these severe measures trial should be made of the effects of electricity, which if it does not actually cure the condition, can, at least in many instances, bring about a considerable amelioration of the symptoms. Bergonié recommends a large electrode to be applied, covering half the face, to the form of which it is exactly moulded. The positive pole is applied to this part, and currents of considerable itensity, 30 to 80 milliampères, are employed. Some patients cannot endure such strong currents, and in these cases longer sittings are to be given. Thus currents of 3 to 12 milliampères may be used for an hour or more at a time with good effects. The treatment should be carried out every day, and as far as possible at the same hour each day. The course may have to be prolonged for as much as three months. Relapses may occur, but they are seldom or never so violent as the original paroxysms. Zimmern (*La Presse Médicale*, April 11, 1903).

BITES BY RABID DOGS.—When a person has been bitten by a dog about which there is any suspicion as to the presence of rabies, the animal should be kept under close observation for the next few days. If it do not develop symptoms of rabies within three or four days of the injury, there is no danger of the patient contracting hydrophobia, as the saliva of the dog is not infective till twenty-four to forty-eight hours before the onset of symptoms. If the dog has disappeared, then it is well to have recourse to Pasteur's prophylactic inoculations at once. If the animal has been killed, the only sure method of ascertaining the existence of rabies is by inoculation of laboratory animals (rabbits) ; but the incubation-period is so long that the result of such experiments cannot be awaited, and protective treatment should be adopted at once. Cauterisation of the wound can never be so immediate as to be of much use. The most dangerous places for a bite from a rabid animal to be situated are the head or neck, since the virus appears to travel to the central nervous system through the peripheral nerves, and the route is shortest in these cases. Authorities vary as to the percentage of all cases bitten by rabid animals which develop hydrophobia. In any case it is not a very large one, probably 25 to 33 per cent. By Pasteur's treatment this mortality is reduced to 1 per cent. Dogs are responsible for 93 per cent. of all cases of infection, cats producing 6 per cent., and other animals 1 per cent. (*Journal des Praticiens.*)

PERSISTENT DIARRHŒA IN TUBERCULOSIS.

℞.	Ichthoformi - - - -	ʒ ss.
	Tannalbin	
	Bismuthi subgall - - -	aa. ʒj
	Codeinæ - - - -	gr. iss.
	Olei menth. pip. - - -	♏. iss.

Misce et divide in chartas VI. S. one powder to be taken every three to six hours, according to instructions. (*International Clinics.*)

EPISTAXIS.—The latest approved method is to use a five-per-cent. solution of suprarenal extract directly upon the mucous membrane. This will at once blanch the mucous membrane and stop the hemorrhage. When the epistaxis is caused by a cardiopathic condition in old persons, the suprarenal extract should not be used. (*Medical Record.*)

CLINICAL USES OF ADRENALIN. — Breton (*Gaz. des Hôpitaux*, June 30, 1903) writes as follows:—"We have employed the following technique (for hæmorrhoids). The patients were such as had hæmorrhoids which had prolapsed or were irreducible, and which gave rise to abundant hæmorrhage at each stool. Fifteen to twenty minutes before defæcation, the patient introduced into the anus (but not into the rectum) a tampon of absorbent wool impregnated with a solution (of adrenaline) of a strength of 1 : 1000. After the first application the hæmorrhage stopped. By continuing this treatment just before defæcation for five or six days we entirely checked the loss of blood and prevented the prolapse of some of the venous nodules. For hæmorrhoids more highly situated adrenaline incorporated in suppositories has given us excellent results. In tubercular hæmoptysis a solution of 1 : 1000, taken in doses of 15 to 20 drops, has often proved successful."

INSOMNIA IN CHILDREN.—In *The Medical Times* for February, 1903, Dr. Reynolds writes as follows with regard to insomnia occurring in children:—"The condition may be the result of too frequent nursing, or due to improper physical condition of the mother, or to some peculiarity about her milk. With bottle-fed children the insomnia may be traced to the bottle or its contents. Should the insomnia continue after these precautions have been taken, a teaspoonful of lime-water or of Vals water may be given between consecutive nursings, and as a laxative a teaspoonful of syrup of chicory, or a pinch of magnesia in sweetened water. If insomnia is due to premature weaning it will usually disappear if the child is restored to the breast. If weaning has occurred at the proper time the regulation of the diet will go far toward relieving any tendency to insomnia. When insomnia is dependent on digestion in children two years of age and upward, a dose of wine of rhubarb or of wine of pepsin at suitable intervals is indicated."

THE PRACTITIONER.

SEPTEMBER, 1903.

IS THE URBAN HOSPITAL TREATMENT OF EXTERNAL OR SURGICAL TUBERCULOSIS JUSTIFIABLE?[1]

By A. H. TUBBY, M.S., F.R.C.S.,

Surgeon to the Westminster Hospital; Senior Surgeon to the Evelina Hospital for Sick Children, &c.

WE are all agreed that the object of the treatment of disease is cure, as quickly and as painlessly as possible, and if this be effected, it should be of such a nature as to enable the patient to earn his or her living subsequently; and, further, the cure should, in the case of an infective disease—and tubercle may be fairly considered such—render him a healthy member of the community, and, *ipso facto*, no longer a source of danger and infection to others.

The question at issue is whether the treatment of tubercular disease of the bones, joints, glands, and mucous membranes, which we for convenience designate by the term surgical or external tubercle, ought or ought not to be undertaken in the hospitals of cities and towns; and if so, does treatment carried out in these places fulfil the criteria which have been mentioned?

In order to examine this question I have been at the pains to collect statistics for the year 1901 from four Children's Hospitals in London and from one in the country. In response to my appeal to the London hospitals, four alone replied, namely:—The Belgrave Hospital, the Evelina Hospital, the Paddington Green Hospital, and the Victoria Hospital.

I was enabled to secure returns without difficulty from one country hospital, the Sevenoaks Hospital for Hip Diseases, so that it has been possible to obtain a comparison between the results of treatment in town and country; but it must be clearly stated that the conditions which obtain in the two groups of hospitals are not identical. A children's hospital in London is maintained for the treatment of all kinds of disease, and the natural wish of the administration is to obtain as many satisfactory results as may be in a given period. Therefore, acute cases of every kind of disease, capable of rapid treatment, are often preferred to more chronic ones, such as the tubercular; and even if the latter are admitted, it is more often with a view to immediate operation, and when operated upon, such cases are frequently sent out before they are fully healed, so as to make room for more acute, and to many people more interesting cases. In fact, the stress and strain upon children's and general hospitals in London is such that it is not possible to give that adequate and prolonged attention to tubercular disease of the bones and joints, which is an essential factor in the success of these cases. Presuming that they could be retained for a considerable time, there is no doubt that the air of a London hospital is not the best for them. Often I have noted a temporary improvement in a child's condition after admission, due to rest, good food, and cleanliness, and for a week or two there has been seen some betterment resulting from the removal of a breaking-down focus of disease; but if such cases are retained for any length of time, the signs of improvement disappear, and they steadily retrograde. Then, as a desperate resource, they are sent off for a short time to the country or seaside, where they may or may not heal, and where they are a focus of infection to other patients just recovering from acute illnesses. Eventually they drift back to London, and begin the weary hospital round over again.

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[1] A paper read before the Society for the Study of Disease in Children at the Brighton meeting, June 2, 1903.

Now, in a hospital entirely devoted to the treatment of surgical tuberculosis there are no conflicting interests arising, nor other and more acute cases to be considered, and therefore everyone works with one object in view, namely, the cure of cases, however chronic they may seem. They are taken in with this object alone, and are retained until they are well, or it is evident that they are entirely beyond relief by any of the present means in our power. Herein lies the essential difference between hospitals where mixed cases are taken in and those devoted to one object alone. The need of the latter is a radical defect in our arrangements.

In compiling the statistics enquiries were directed to the following points, and the result has been carefully tabulated :— The age and sex of the child have been noted, and in this paper children exclusively are dealt with, namely, boys under the age of 10 years and girls under the age of 12 years. Then the duration of treatment previous to the last admission into the hospital from which the returns are made has been ascertained; also the number of admissions to other hospitals, because we find that many children go from hospital to hospital, while some few return again to the same hospital. The duration of the stay in the hospital on the last occasion is also given, with the total number of operations performed from first to last, and the results under three headings :—

1. *Relief.*—Meaning by this that the patient's condition has been somewhat ameliorated by a stay in a hospital, but that the disease has not been entirely cured, as is evidenced by discharging sinuses, infection of the skin, or the incidence of other tubercular lesions.

2. *Cure.*—That the disease has subsided and healed, and that there are no signs of active tubercle anywhere; and further, that there is a reasonable probability of the child being able to gain a livelihood, or, at any rate, that he has ceased to be a source of further infection to himself, or a focus of danger to other people.

3. *The Number of Deaths* has been noted in the hospital. The numbers of these given must be necessarily smaller than actually occur, because many of the so-called "relieved" cases are lost sight of, and a fatal result follows eventually from phthisis or tubercular meningitis. Too often the child lingers on, and dies at home, having spread infection far and wide, or drifts into a hospital, the statistics of which have not been available to us.

To refer to actual details. We find that in the urban hospitals mentioned, 218 cases were admitted, being 115 females and 103 males; they were of the average age of 5 years and 8 months—a very tender age to be the victims of chronic trouble such as this—and they had undergone treatment for 16 months previously. Forty-five were afflicted with tubercular glands in the neck; 10 had tuberculosis of the skin, 90 of the joints, and 64 of the bones. The minimum stay in the hospital in London was 5 days, and the maximum 176, the average being 41 days, or nearly 6 weeks. On these 218 patients 415 operations were done; so that, taking the average, about two operations were performed on each patient; and in one case it is recorded that he was operated on as many as 18 times, another 12, and another 11. These operation-statistics constitute, to my mind, a very strong indictment of the urban hospital treatment of tubercle. It is a moot point to me whether operations on the bones and joints of children in city hospitals for tubercle are not productive of as much harm as good, in the way of further spreading the disease by infecting fresh tissues. However, as the result of all this expenditure of labour and time, only 68 of the 218 cases, or 31·2 per cent., were cured; 128, or 58·6 per cent., were relieved—and relief is no cure in tubercle, for half-cures are worse than none—in 13 cases the result was doubtful, or unchanged, and 9 patients are known to have died in hospital.

Now we turn to the statistics of a country hospital, the Hip Hospital at Sevenoaks; and here it must be premised that under the present chaotic arrangements for the treatment of tubercle, this hospital does not have a fair chance with all the cases from the commencement, and this is, as I shall insist upon later, an essential point. A considerable number of their patients are sent down from London hospitals after operative treatment has been tried and failed. In one case three operations had been done, and the remark in the note-book is to the effect that "the result is not one to be proud of." I need not weary you with the repetition of all the statistics, but give merely the salient points. Forty-two cases were under treatment in one year: the average age was a little over six years. Thirty were cases of joint-disease, 10 of the bones and spine, and 2 suffered from enlarged glands. The average duration of their stay in hospital was 10½ months. The total number of operations was 3, and the results show 14 cures; 24 were relieved, and many of them are progressing most satisfactorily, and promise thoroughly well. Three were discharged incurable, and one died of tubercular disease of the kidney.

Now, it will be noted that in the case of this hospital the duration of stay was 45 weeks, as compared with six weeks; the proportion of cures was greater than the London Hospitals can show; while the operations performed were represented by the small number of three; that is to say, the percentage of operations in the town hospitals, as compared with that in the country hospital, is as 19 to 0·07, or nearly thirty times as many.

There can be no doubt as to the conclusions to be drawn from these results.

1. *As to Cure.*—The number is by no means so great in town as in the country, nor is the cure so complete, for the number of relapses is far greater, as is shown by patients continually going the round of the hospitals. The urban hospital treatment cannot, therefore, be economical—meaning by this term that the best results are being obtained for a given expenditure of time, money, and energy. Under the present disjointed arrangements, whereby country hospitals are compelled, from sheer pity, to take the cases that have failed in London hospitals, the resources of both are wasted. It is true that cases remain longer in hospital in the country, but the results are infinitely better.

Then we come to the question of the painlessness of the treatment. There can be no doubt on this point, when we contrast the number of operations, and I am disposed to say that the enormous number of operations done in London, as compared with those in the country, constitutes a very grave state of affairs, and needs urgent consideration. For, after all, it must be remembered that many of the operations on bones and joints for tubercle are very far from being such successes as they promise when they are undertaken. There are no operations which need more careful consideration as to the after-possibilities than those for tubercular disease of the bones and joints. Everything looks so well on the operating table, and it appears as if the disease has been removed, but the disappointments are many and great. More particularly is this so, after excision of the hip and knee. We can all call to mind a large number of limbs shortened or twisted, and therefore useless, and with many recurrences of tubercle in the soft parts, followed by sinuses and scrapings. Above all, we meet from time to time cases of hip-joint trouble in which it is necessary to amputate; and it must surely seem that the removal of a fourth part of a patient's anatomy for a curable disease is, under any circumstances, a grievous reproach to modern surgery. After considerable experience, I cannot help expressing the opinion that better results are obtained in those cases which are treated by rest from the first, and where there is no excision or ablation of bone and wide cutting of the soft parts.

We will now pass on to the suggested remedies.

1. *The Place of Treatment.*—Tubercular children should not be put in hospitals in cities or large towns, but properly-equipped establishments should be started in the country or at the seaside. The increasing means of communication will easily meet the difficulty of the attendance of competent surgical officers.

2. These hospitals should be devoted to one object only, just in the same way as sanatoria exist for the open-air treatment of phthisis alone. If internal tubercle is treated in special hospitals, why not external?

3. The rural hospitals must be conducted on definite principles. There should be no limit as to age, the younger children being equally eligible with the older. In fact, the younger the child, the more hopeful is its case, and the more urgent is it to take it in hand at once and thoroughly. Cases once admitted should stay until they are cured, or clearly proved to be incurable; and for the latter separate establishments should be provided, so that they cease to be a danger to the community. Cases which are undergoing cure should not be liable to interruption, owing to the want of subscribers' letters or other red-tapeism, and a case once entered upon must be followed right through.

Now to obtain this ideal state of affairs, the present chaotic and wasteful absence of system must be abandoned. The first essential is to have a central council to deal with the whole matter and with cases as they arise. Every case applying for relief to a city hospital should be notified to the central council, who also administer the rural hospitals, and the cases should be placed in some one or other of them, careful notes of the progress being made. And after the child has left the hospital examination should be carried out from time to time; and if recrudescence occurs, the patient should be sent back to the particular hospital at which he was treated, and as far as possible placed under the same surgeon, with the object of securing a continuity of treatment under one skilled observer—a most important factor in success, the absence of which is so striking at the present day. Until a central board is established with powers such as have been mentioned, it is to be feared that external tubercle will go on increasing.

An important question is as to funds. While some hospitals have their own convalescent homes, where they mix indiscriminately tubercular and non-tubercular cases, many others have none. It is found on enquiry that one child in every ten admitted to a general children's hospital is the subject of external tubercle. Surely it would be better and more economical for such hospitals to close one-tenth of their beds, and earmark one-tenth of their income for the purpose of establishing branch hospitals. At any rate they would turn over the remaining nine-tenths of their beds quicker with the more acute cases of disease. If it should happen that the amount which each hospital can contribute is too small to establish a separate branch hospital, then there is no reason why several committees should not combine, and have beds proportionate to their contributions.

But the gist of the whole matter appears to me to be the need of a central and organising committee, all the members having an intelligent appreciation of the drawbacks of the present arrangements, and an earnest desire to tackle the question in a thorough and businesslike manner.

THE TREATMENT OF GOUT IN CARLSBAD.

By B. LONDON, M.D., *Carlsbad.*

(*Continued.*)

TREATMENT.

THE lines upon which the therapeutic treatment of gout must be undertaken are indicated by our knowledge of the hyperacidity of the tissue-juices, and of the retardation of metabolism produced by uric acid.

In the treatment of arthritis, *prophylaxis* must claim our foremost attention. In consequence, our regulation of the diet is almost pedantic, and we take care to avoid excess in food and drink, only allowing an unirritating dietary, regulating the bowels, advising work and bodily exercise, so as to increase the circulation and favour excretion of waste products. And in improving metabolism, the effect of Balneo- and Hydro-therapy is unquestionably most favourable. The avoidance of alcoholic excess, or of lead-intoxication, is of primary importance.

To avoid overloading the organism with uric acid, we endeavour not only to prevent its formation, but also to aid its excretion, to accelerate its further oxidation, and lastly to increase its solubility in the blood and tissue-juices.

Great care must be taken in the regulation of the diet to reduce in amount the antecedents of uric acid contained in food. This can be effected, as we shall see later, by drugs such as quinic acid, salicylic acid, antipyrin, &c.

Recent investigations by Krüger, Petren, Schittenhelm, and Weintraud have shown that the daily excretion of purin-compounds, in the form of bases and nuclein-bases, is nearly three times greater in the fæces than in the urine. We may, therefore assume that purgatives exert an important influence on the formation of uric acid, and that by this means the excess of purin in the organism may undergo certain changes.

The important part played by the digestive apparatus in arthritic processes, and the various relationships which exist between the changes occurring in the intestinal contents, as well as in their walls, and uric-acid metabolism, justifies the assumption that we may influence the course of gout through the agency of the alimentary canal. We, therefore, commonly prescribe a saline aperient (in commencing a "cure") to those patients to whom indigestion has induced or accompanied an attack of gout, more especially if habitual constipation is present.

Uric-acid excretion is produced by encouraging renal secretion by the copious administration of fluids, or by means which influence the circulation of the blood. An endeavour may be made to raise the uric-acid elimination by oxygen-inhalation, by the ingestion of thyroid extract or by the use of spermin.

Since uric acid is deposited in the gouty areas in a difficultly-soluble form, we endeavour to remove these deposits by inducing more favourable conditions for their solution. An attempt has been made to raise the alkalinity of the blood and tissue-juices by the administration of alkaline carbonates and alkaline salts of vegetable acids; but it is found that the excess of alkalies is thrown out in the urine. We tried, further, to facilitate the solubility of uric acid in the body by giving basic salts, such as lithium, which, in a laboratory experiment, will form easily-soluble salts with uric acid. It was thought that the same experiment would succeed in the organism. This has, however, not been verified. No greater success attended the administration of organic nitrogenous bases, such as piperazin, lysidin, &c., than was obtained with inorganic salts. They are incapable of dissolving the uric acid in the body. It is to be hoped that we may be able to modify the action of uric acid in the body by producing a firm combination between it and other organic atom-complexes, as it is highly probable that such complicated combinations of uric acid with other substances are of the highest importance in modifying uric-acid metabolism in the organism. Recent researches show that uric acid enters into loose combination with formaldehyde. On the production of this diformaldehyde uric acid depends the action of urotropine in the treatment of gout and bladder-diseases. Experience proves that urotropine, in the body, liberates formaldehyde, which is able to take up a portion of uric acid in the form of the easily-soluble diformaldehyde uric acid.

Experience teaches us that suitable dieting has achieved greater results than any measures in use up to the present, which aim at bringing about a solution of uric acid. During the decomposition of the food in the body many acids and bases are produced; and the relative alkalinity of the juices is modified by the food introduced, owing to its containing preformed salts. It is also possible that in the decomposition of the food, many organic bodies are evolved, which are able to enter into combination with the uric acid. We attach some importance to the belief that the disintegration of nucleic acid may be responsible, not only for the formation of uric acid, but also for its solubility and diffusion in the body; and it is possible that this may be dependent upon the composition of the food.

For the successful treatment of arthritis, we must attend to the individual characteristics of the disease, and deal with them symptomatically. We are accordingly presented with the treatment of an acute attack and its immediate sequelæ. Then comes the treatment of arthritis as a general disease—the uric-acid diathesis. Lastly, the chronic affections produced by gout have to be considered.

In the first place, we must impress upon our patients the necessity of avoiding everything that experience teaches us is likely to precipitate an attack, such as excesses in eating and drinking, exposure to cold, and over-fatigue. In the acute attack, local treatment claims our attention. As a rule, complete rest of the extremity is required to relieve the pain; but I was frequently able to diminish the duration of an attack by allowing bodily exercise at the earliest possible moment.

Local treatment must be preceded by absolute rest. Then compresses of linen or gauze soaked in Burrow's solution (Liq. Plumb. Subacet. 25 grammes, aluminis 5 grammes, aq. dest. 500 grammes) must be applied, covered over with cotton-wool and changed every two hours. Great relief may be obtained, before the application of the wet compresses, by gently rubbing in a mixture of equal parts of mesotan and olive-oil. Or the parts may be frequently painted with a 40-per-cent. solution of ammonium sulpho-ichthyolate in spirit of æther, and alternating this with the compresses of liq. Burrowi every two hours.

Cotton-wool dipped in alcohol (60 per cent. to 70 per cent. strength) covered by oiled silk, to prevent too rapid evaporation, and applied to the affected part, sometimes acts admirably.

Since the inflammation, in an acute attack of gout, depends upon the accumulation of an abnormal amount of uric acid in a state of combination, it is the function of therapeutics to reduce this excess of uric acid, and then to convert the salts into a form capable of excretion. Colchicum and its preparations fulfil the first indication. Colchicum possesses the property of restoring to the tissues their diminished capacity for decomposing uric acid, thus getting rid of the latter, and, with it, the cause of the pain and inflammation. Colchicum further influences the renal secretion as well as intestinal activity, diminishes pain, and, lastly, is said to modify the abnormal cell-functions which underlie the gouty metabolic disturbances.

I usually employ the Tinct. Colchici (·5 gr. to 1 gramme for a dose, 6 grammes per day), or the Vinum Colchici ♏. 10 to 20 several times a day, up to ♏. 100 per day. At the present time, many gout-specialists on the continent prefer pure colchicine. Here in Carlsbad, colchicine pills are generally prepared from colchicine prepared by Merck (Darmstadt), and I prescribe three or four doses of one milligramme; one pill every half hour or hour.

In cases of some duration, I give a pill three or four times a day (℞. colchicinæ 1 centigramme—fiant pilulæ decem.). Care must be taken in the administration of this drug. The extraordinary success obtained by the Liqueur Laville in acute cases, is due chiefly to the fact that it contains large quantities of colchicum.

The recently much vaunted preparations of quinic acid are said greatly to reduce the excretion of uric acid in the urine. These are quinate of lithium (urosin), quinate of piperazin (sidonal), quinate of urotropine (quinotropine), and quinic acid anhydride (neusidonal), but they cannot compete with colchicum in its rapidity and certainty of action.

To promote the excretion of uric acid, and at the same time to render it capable of excretion, many other organic acids have been found of use, particularly salicylic acid and its preparations. Recent investigations confirm the already well-known fact that these not only increase the nitrogenous output, but also the uric-acid excretion.

The chief effect of the salicylates in gout, as in rheumatic affections, is to relieve pain ; but at the same time they also cause a diminution in the swelling. Of the preparations of salicylic acid, aspirin, in doses of one gramme hourly, four to five grammes at night, has stood me in good stead.

In troublesome cases where no definite effect could be produced with aspirin in the course of a few days, I am in the habit of substituting antipyrin ($\frac{1}{2}$ to 1 gramme thrice daily), salol, salipyrin, or phenacetin (in the same doses as antipyrin) on account of their anodyne and hypnotic properties.

If the acute attack persists in spite of internal and external treatment, I have found a combination of iodide of potassium and colchicum of great use (℞. Pot. Iod. 5 grammes; Tr. colchici semin., 10 grammes; aq. dest. 150 grammes :—one tablespoonful thrice daily). This mixture should be continued, in spite of improvement, for six or eight days, in daily-diminished doses.

As soon as the pain is even slightly relieved, I allow the patient to leave his bed, and spend the day on a couch. Walking exercise is encouraged as early as possible. This treatment will much shorten an attack that would last for weeks were the patient kept in bed too long. If the upper extremities are involved, I order suitable exercises later on. The last traces of swelling and stiffness disappear only with constant use of the affected part.

Our natural thermal baths (*Sprudelbäder* and *Moorbäder*) which have been proved to bring about the presence of free uric acid in the urine, effect a cure of the lingering traces of an acute attack in a surprisingly short time ; so that crippled patients are able to get about quite well after a few baths. The often strikingly-favourable effect of the thermal baths on pain and swelling is due to the fact that, in the acute stage, the uric acid which is excreted in a combined state, rapidly passes into the free form.

When joint-attacks are complicated by conjunctivitis, keratitis, iritis, glaucoma, migraine, lumbago, bronchitis, laryngitis, pneumonia, skin-diseases, &c. of gouty origin, in addition to anti-arthritic measures and diet, treatment must be directed to the local phenomena, wherever indicated.

With regard to the diet in an *acute* attack, only such food should be allowed as is incapable of producing free uric acid, and this requirement is essentially fulfilled by the carbohydrates. These are prescribed in easily-digestible form, such as rice, pearl-barley, oatmeal soup, purée of albumen-containing vegetables. Since vegetable albumen contributes little or not at all to the formation of uric acid, we may freely allow aleurone bread. Fruit, with the exception of strawberries and raspberries, mostly cooked, may be given in the form of jam. Lemons may be freely given ; the juice may be taken in food, or may be drunk as lemon-squash with some effervescing alkaline table-water.

The white of egg being free from nuclein, and the yolk only containing a paranuclein (vitellin) incapable of separating off purin-derivatives, enables us to prescribe eggs *ad lib.* Milk being very nourishing and unirritating, and possessing high diuretic properties, is found most useful for gouty people, but need not be exclusively prescribed. We avoid meat and meat-soups, at first, entirely.

The stimulating action of alcohol on the blood-vessels, produces a hyperæmia of goutily-affected parts, thus increasing pain and inflammation. It also much diminishes the capacity of the tissues for decomposing uric acid; hence, in the acute stage, alcohol must be prescribed as little as possible. Many arthritic patients, in time, find themselves much better if they give up taking spirits altogether. If stimulants are absolutely necessary, half a pint of light Bordeaux or a couple of table-spoonfuls of whiskey, both drinks freely diluted with water, may be taken. For the tiresome thirst that occurs, a good ordinary water may be given, or one not too alkaline, such as Apollinaris, Seltzer, and in Carlsbad, Biliner or Neudorfer. The strongly-alkaline waters, like Vichy, Fachinger, &c., are to be avoided in the acute stage, as their richness in carbonic acid may lead to a precipitation of sodium biurate in the tissues.

No alkaline medicines or alkaline purges, such as Carlsbad salts, must be given in the acute stage. Instead we may use castor oil, cascara sagrada, rhubarb, glycerine capsules and enemata.

In the general treatment of the gouty (uric-acid) diathesis, we must gradually accustom the patient to a different mode of life. The treatment consists principally in living very regularly, and in increasing general and local metabolism, which is always an imperfect one in such subjects.

Next in importance to careful dieting is a rational mode of life. Instead of a sedentary indoor existence in which want of occupation is occasionally varied by unusual mental effort, exercise in the open air should find a place, the effect of which on digestion, metabolism, and circulation will favourably counteract the tendency to uratic precipitation. Horse-exercise, bicycling, hill-climbing, and methodical gymnastics should form part of the day's work. In elderly people or in those in whom the disease has made progress, rubbing or kneading the joints or the whole body may be substituted for these exercises. To these may be added hydrotherapeutic measures, particularly rubbing down the body with salt-water, as well as the thermal treatment mentioned below. Travelling in a suitably-selected climate sometimes brings about marked improvement.

Dietetic treatment consists in reducing the amount of food, as compared with the quantities usually taken. In this respect we must be guided by physiological principles, and the food-stuffs can be conveniently considered under the heads of proteids, fats, and carbohydrates. The diet must be accurately prescribed, the amount and character of the meals, and the hours at which they should be taken being carefully specified. The preparation of the food must be simple, particularly in the case of the meat-dishes described below, without rich sauces or condiments which serve only to irritate the stomach and kidneys. The use of coffee and tea must be considerably curtailed and in some cases absolutely forbidden.

If alcohol is not a direct ætiological factor in gout, we cannot deny that the ingestion of alcoholic drinks acts prejudicially on the course of the disease, particularly in the frequency and intensity of the attacks. It is therefore better for the patient to eschew alcohol. Water is unquestionably the best drink for arthritic patients. Fruit and green vegetables are very much to be recommended on account of their richness in vegetable acids and salts; they increase diuresis and combine fully with the uric acid in the urine. Vegetable acids, such as citric, tartaric, and malic acids, are converted in the body into carbonates and appear later in the urine in the form of alkaline carbonates. Sour milk is contraindicated owing to the presence of lactic acid, which is harmful; on the other hand sweet fresh milk markedly reduces the excretion of uric acid and diminishes the amount of free uric acid.

The ingestion of carbohydrates must be limited to the smallest amount, but one must individualise in this respect. If we want to reduce the body-weight, we must exclude amylaceous foods; if the nutrition requires to be improved, then we have recourse to easily-digestible carbohydrate-containing aliments. Fats, in the form of fresh butter, will be only prescribed in special cases and in amount suitable to the digestive capacity of the patient concerned.

Besides mechanical and dietetic treatment, a third form of medication is indicated, since by means of the two former, it is difficult to convert the uric acid completely into the combined form. For this purpose alkalies are preferred. It is generally accepted that alkalies favourably influence gout in its various stages and particularly in its local manifestations. However we may explain the increase of uric acid in the blood, it is theoretically quite intelligible that the introduction of innocuous salts, which increase the alkalinity of the body-juices and reduce the acidity of the urine, must increase the capacity of the tissue-juices for dissolving uric acid, and so diminish uratic precipitation. Further, these alkaline salts are well known to affect the functions of the alimentary canal very favourably, and some of these alkalies have been shown to produce a direct increase in nitrogenous interchange. This explains why it is sometimes possible to postpone an expected gouty attack by the administration of alkalies, and to banish the dyspeptic troubles and other antecedents of gout. The favourite, and according to experience, the most effectual method of administration of alkalies is by means of mineral waters which contain the salts that we are here specially concerned with, viz., potassium carbonate and sulphate, sodium chloride and other sodium-compounds in suitable forms. At the same time alkaline waters ought to be sparingly taken, so as to avoid overloading the body with alkalies, since an excess of alkali (especially bicarbonate and carbonate of sodium) may directly induce a deposition of biurates in the tissues. Of the numerous springs to be mentioned later, the selection will depend, in a given case, upon the constitution of the patient, the stage of the disease, the intensity of the associated internal affections (particularly where the alimentary canal is involved), and the existence of such complications as obesity, diabetes, &c. Up to the present, the most suitable are the simple alkaline waters, the earthy or calcareous waters, the sulphated alkaline waters, and the common salt waters.

The therapeutic value of mineral waters depends partly upon the water they contain. The flushing of the tissues must surely contribute to the solution and excretion of urates. Besides this, digestion, metabolism, and renal secretion must be favourably influenced by the measure of the salts. The indications for the prescription of the various mineral springs must necessarily depend upon practical experience. And experience teaches us that the table-water most suitable for daily use by the arthritic patient is one which contains a small amount of salts, such as the simple and alkaline waters, the earthy mineral waters or the lighter common salt waters. Such are :—Apollinaris, Bilin, Contrexéville, Gerolstein, Gieshübel, Evian, Cinciano, Fachinger, Kronthal, Krondorf, Nocera, Salvator, Sulzmatt, St. Galmier, Vittel, Wildungen.

The stronger mineral waters are used in special cases, and generally combined with thermal baths, and the cure is generally undertaken only once or twice a year, for from three to six weeks, if possible at the source itself. In selecting a spa, not only must the composition of the waters be taken into account, but also the scenery, climate, conditions of life, and other adjuvants to the "cure;" and last, but not least, regard must be had for the proved and acknowledged capacity of the physicians practising there. In these respects every case must be considered individually.

We have learned empirically that such gouty patients as suffer from slight dyspepsia, are not over-stout, and are inclined to diarrhœa, derive most benefit from the alkaline springs of Neuenahr, Vichy, and Vals. Where, however, much sand and gravel are passed, and the case is complicated by disease of the urinary passages, the alkaline earthy springs of Contrexéville or of Wildungen are to be preferred.

Gouty patients troubled with gravel, who are also the subjects of albuminuria, of congestion of the portal system, of enlarged liver, with more or less jaundice, are likely to be very much improved by employing the sulphated alkaline waters of Carlsbad, Bertrich, Strathpeffer, or Tarasp. Marienbad is indicated in the case of obese patients of robust constitution, especially if they suffer from chronic constipation and hæmorrhoidal troubles. If such patients, however, are of weakly constitution, and are likely to become cachetic in consequence of long-continued disease, it would be better for them to go to Homburg or Kissingen.

Ems is useful for cases of chronic bronchial catarrh, and Nauheim for heart-disease in gouty subjects. Where there are evidences of stiffness in the joints, chronic enlargements, and uratic deposits, good results may be obtained by taking the common salt springs and baths of Wiesbaden and Baden-Baden, or the sulphur-baths of Aix-la-Chapelle, Aix-les-Bains, Baden in Austria, Baden in Switzerland, Schinznach, Weilbach, Nenndorf, Dribourg, "Fango" baths in Battaglia, or the natural vapour-baths in the grottoes of Bormio and Monsummano. Should such patients be weak or badly nourished, such places as Buxton, Bath, Gastein, Plombières, Ragatz-Pfaefers, Teplitz, Poestyen, Wildbad, Mehadia, Herculesbad are preferable. Before sending patients to these spas, where, in addition to water- and bath-cures, the most varied and modern physical appliances are brought into requisition, we must carefully investigate their general constitution, age, and possible complications.

The essential factors in the general treatment of the gouty diathesis are the warm baths, which, in conjunction with the water-cures and subsequent use of physical methods, are mostly sought after and used by gouty patients. Good results have been obtained by the use of electricity applied locally. Arthritic deposits in the sheaths of the extensor tendons have been frequently beneficially treated by galvanism. Recently, on the recommendation of Edison, cataphoresis with lithium has been tried. This has either been done by passing the galvanic current through the affected joints or tophi, by means of electrodes dipped in a concentrated solution of carbonate or chloride of lithium; or by means of electric baths. In this manner rapid diminution of pain and reduction in the swelling of gouty finger-joints have been brought about.

THERAPEUTIC TREATMENT OF GOUT IN CARLSBAD.

As is well known the alkaline muriated-sulphated waters of Carlsbad owe their healing properties chiefly to the combination of sulphate of sodium with sodium carbonate and sodium chloride. After absorption of the constituents of the Carlsbad waters, there is a relatively-rapid disappearance of the sulphates from the stomach; they are discharged for the most part in the fluid evacuations. The carbonates are demonstrable in the urine, which is thereby rendered alkaline. If excessive quantities of sodium carbonate are introduced for any length of time into the body, it loses its sodium chloride. It is, accordingly, of the highest significance that the innocuousness of the Carlsbad waters is due to the fact that they are at the same time so rich in sodium chloride that they can, of themselves, make good the deficiency, apart, of course, from the value of common salt in favouring gastric secretion. Not only do the alkalies contained in the Carlsbad waters facilitate the solution of uric acid in the body-juices, but they are especially valuable in increasing metabolism.

Experience teaches us that the use of hot-air baths, hot-water and vapour-baths, and the drinking of waters increases metabolism, thereby raising uric-acid excretion, and bringing about a gradual disappearance of even considerable and long-standing gouty deposits.

To the two properties of the Carlsbad waters so valuable in the treatment of gout, viz., alkalinity and a high temperature, must be added a third; that is, an eminently favourable influence upon the digestive tract. By rapidly flushing out the alimentary canal, in which peristalsis has been excited, a large quantity of undigested food is got rid of; thus removing a portion of the material responsible for the production of uric acid. This may be carried so far that the body has to part with some of its own fat, sometimes even of its own albumens, in order to maintain the metabolic balance. Owing to these marked qualities, alkalinity, high temperature, and purgative action, the waters of Carlsbad are recognised as the medication, *par excellence,* in the treatment of gout.

The majority of gouty patients whom I have an opportunity of treating here suffer, in consequence of disturbed metabolism, from chronic constipation with its attendant troubles (auto-intoxication by products of stagnation in the intestinal contents—ptomaines), feeling of lassitude, dislike for work, loss of appetite, migraine, concentrated urine, flatulence, &c. In these cases I nearly always begin with a saline purge. Of course, the Carlsbad waters and other adjuvants must not be prescribed in a stereotyped manner, considerable discretion being used in individual cases. We mentioned above that, other things being equal, uric acid is relatively very soluble in hot alkaline fluids; that its solubility varies directly as the temperature of the blood. The various warm springs in Carlsbad vary from 102° F. (Kaiser Karl Quelle) to 162° F. (Sprudel), thus raising the temperature, increasing the heart's action, and raising the blood-pressure considerably. I have noticed that with the same quantities of thermal water, the hotter it is, the better the results. A rise in the tension and frequency of the pulse associated with a corresponding sensation of inner warmth is at once noticeable. The thermometer shows a rise of ·2 to ·4 degrees in patients drinking the Sprudel in the morning before breakfast.

The appearance of changes in the circulatory organs and kidneys, which are almost pathognomonic of gout, at once suggest a reduction in the quantity, temperature, and duration of the Carlsbad waters and baths prescribed. I have, however, been taught by experience that patients soon become accustomed to the hot springs, if the cure is commenced with the cooler waters, the temperature being then gradually increased.

Gout, more than any other disease in Carlsbad, requires treatment by the hottest springs, and in the largest possible quantities, even if these have to be distributed over the whole day. For instance, in the early morning, fasting, we order three glasses at intervals of a quarter or half an hour (having regard to each individual case). Then breakfast an hour later. At 10 or 11 a.m. another glass, either at the spring or taken at home. Dinner at 1 p.m. From 3 to 5 p.m. walking-exercise, especially gentle hill-climbing. Carlsbad is surrounded by a series of hills and mountains, which might have been created specially for the Oertel treatment. These are intersected by miles of systematic and well-designed paths, in the most beautiful country, constituting an element the value of which cannot be over-estimated in the treatment of obesity and heart-disease which so often complicate gout.

At 5 p.m. another glass of the prescribed spring. Supper at 7 p.m. Should constipation still persist, in spite of these five glasses of water, 5 grammes (a heaped teaspoonful) of Sprudel salt is added to the first early morning glass; or a glass of cold Felsenquelle may be taken at bedtime or immediately on rising.

The springs mostly affected in Carlsbad are:—

(1) Sprudel (166° F.); (2) Franz Josef Quelle (149° F.); (3) Bernhardsbrunn (148° F.); (4) Neubrunn (145° F.); (5) Felsenquelle (140° F.); (6) Theresienbrunn (140° F.); (7) Mühlbrunn (133° F.); (8) Schlossbrunn (130° F.); (9) Marktbrunn (122° F.); (10) Kaiserbrunn (120° F.); (11) Elizabethquelle (116° F.); (12) Kaiser Karl Quelle (102° F.).

We know from experience what an important part is played by the baths in the successful treatment of articular gout. For this purpose local and general baths are given. These are:—(1) Sprudel baths, or mineral baths; (2) peat-baths; (3) pine-baths; (4) electric-light baths; (5) Russian and Turkish vapour-baths; (6) modified hydrotherapeusis.

Lastly, there are at Carlsbad two medico-mechanical Zander Institutes for medical exercises and massage.

To those patients who are affected with heart-disease or arteriosclerosis, and who are unable to stand the general baths, I generally apply heat locally. I find this the best method of producing local hyperæmia, of improving the circulation, and raising the temperature, thus hastening the solution and removal of gouty deposits, while the alkalinity and dissolving-capacity of the blood is at the same time augmented by drinking the waters. Besides, heat applied locally (by means of peat-compresses and hot-air appliances) is always much better borne than are the general baths.

A renewed attack of gout in the joints is very often produced as a result of the thermal water-baths, or peat-baths, or hot-air baths, or even of the local baths, including the hot peat-poultices, by the irritation and congestion induced by the high temperature of the bath on the joints, rendered sensitive by their impregnation with uric-acid crystals, that is to say, an exacerbation of the local inflammatory areas is brought about. These attacks certainly produce in the joints similar sensations to those occurring in true gout. But there is an absence of the typical premonitory symptoms of general disturbance, as well as on the sudden onset in the night, together with the variations in the uric-acid quantities, which are so characteristic of typical gout; sometimes it is impossible to distinguish between the two. In these cases I usually raise the limb and bring it into a more horizontal position of rest; the affected joint is frequently painted with a 30- to 40-per-cent. solution of ammonium sulpho-ichthyolate in spirit of ether; after which from half-hourly to two-hourly lint or gauze compresses dipped in a solution of Liquor Burrowi (Liq. Plumbi Subacetat. 25 grammes, Aluminis 5 grammes, Aq. dest. 500 grammes) are applied and changed every two hours until the disappearance of the inflammation, which usually occurs in a couple of days.

In the true gouty attacks, apart from external treatment, I frequently have to give aspirin or phenacetin, and sometimes colchicum-preparations in order to diminish the pain.

To obviate these attacks, or pseudo-attacks, it is as well to apply these compresses of Liquor Burrowi after each local hot application, as well as after each hot bath; and to do this on the evening of the same day, about from five to eight hours afterwards, leaving them on for a couple of hours. The result is nearly always successful. We are thus enabled to apply the hottest compresses without anxiety, to bring about the greatest amount of hyperæmia required, lasting for several hours, and favouring the dispersion of the deposits. Such compresses put on at night, prevent the development of an inflammation in the predisposed joints. That form of gout which manifests itself by gastric and intestinal symptoms is particularly successfully treated by a cure in Carlsbad. The dyspeptic disturbances, loss of appetite, pyrosis, colicky pains, nausea, vomiting, and diarrhœa, disappear in a relatively short time; the free carbonic acid, common salt, sodium carbonate, and sodium sulphate contained in the waters exercising a powerful influence along the whole of the alimentary canal.

Chronic constipation is not always associated with gout. We frequently see cases of troublesome diarrhœa. In these cases peristaltic action is reduced by drinking the hottest springs; the result being enhanced by rectal injections of Sprudel from a considerable height and at a fairly high temperature. I have noticed in patients suffering from visceral gout, with few exceptions, a painful hypertrophy of the liver, which was not infrequently associated with glycosuria. In these numerous cases of hepatic gout, metabolism is encouraged by drinking the waters combined with the application of local peat-poultices and Burrow's compresses at night, the results being nearly always excellent. Glycosuria must of course be treated dietetically in addition to prescribing the waters and baths.

In recent years I have noticed an increasing number of different forms of dermatitis, particularly eczema, erythema, psoriasis, urticaria, and furuncles, which were intractable to local and symptomatic treatment at home, but which were sometimes improved, sometimes altogether cured, after the treatment of the uric-acid diathesis of such patients had been taken in hand.

Besides the exercises above mentioned, such as walking, riding, cycling, gymnastics, lawn-tennis, and golf, carried on in the fresh pure air of our extensive pine-woods, a strict regulation of the diet is observed, each case being treated on its own merits. The first and great principle of dietetic treatment consists in moderation in every particular. Excess in eating not only increases the production of uric acid, but leads to the production of acid principles which unfavourably influence the later development of uric acid. Moderate eating avoids the excessive formation of uric acid which occurs in gouty patients; digestion is carried on better, and there is less susceptibility to excessive acid-production which occurs when the stomach is overloaded and the digestive processes too often excited. In gouty people the absorption of nourishing principles proceeds much more unsatisfactorily, in spite of apparently good appetite and apparently good digestion, than it does in the healthy person; for the majority of these patients excrete much smaller quantities of uric acid and urea in the urine.

Therefore, in Carlsbad, the patients are only allowed three meals. Breakfast in the morning, dinner at midday, and supper in the evening. All intermediate meals are forbidden. During the intervals of eating, the stomach and intestine recover their power of absorption, and the food is better assimilated.

Before describing the menu (with more or less variations) which is usual in Carlsbad, I propose to mention, in general, the articles of diet and the beverages which we are in the habit of allowing or forbidding according to our experience.

We only allow albuminoids in very small quantities, in the form of meat, which must not contain many extractives, and must be easily digestible, such as fish, veal, and fowls; at rare intervals, a little beef cooked in the form of a *pot-au-feu*. We exclude such organs as liver, spleen, thymus gland, brains, and kidneys, on account of their richness in nuclein. I usually prescribe a maximum of 250 grammes of meat per day. As a substitute I allow albumen in the form of eggs, fresh milk, and cream-cheese. Fat is allowed in moderate quantity in the form of butter, cream, and salad-oil. Carbohydrates, or farinaceous food, are introduced in the form of bread (200 to 300 grammes per day), soups, potatoes (100 grammes a day), and fruit.

To recapitulate:—We allow thin meat-soups, containing vermicelli, sago, rice, barley, oatmeal. Meat we give in the form of veal, mutton, chickens, pigeons, turkeys, young geese, ducklings, partridges, pheasants, lean ham, fresh fish, such as trout, pike, perch, sole, whitebait, herrings, fresh oysters, small portions of crab, small portions of fresh caviare, boiled eggs, cream-cheese, small quantities of Dutch and Swiss cheese, Edam cheese, Cheddar, milk, cream, fresh butter (in small amount), black bread, white bread, biscuit. Small quantities of fresh oil, salad-oil, green vegetables, head of lettuce, spinach, artichokes, endives, watercress, French beans, carrots, fruit, oranges, lemons, apples, pears, plums (the three latter preferably stewed), cherries, strawberries, raspberries. After the Carlsbad cure we recommend grapes, melons, peaches, pineapples.

Beverages:—Pure spring-water preferably, mineral waters, (table-waters mentioned above), weak tea, light wine, Bordeaux (one pint a day). Whiskey, two tablespoonfuls diluted with water at meals; weak *café au lait.*

We forbid condiments, *sauces piquantes,* mayonnaises, *pâté de foie gras,* extracts of meat, sausages, tomatoes, ragouts, sweetbread, brains, liver, kidneys, salmon, eels, sardines in oil, anchovies, lobster, sharp cheese (Cheshire, Roquefort, Gorgonzola, old Camembert), sour milk, butter-milk, koumiss, kefir, *confitures,* sweet milk-puddings, onions, garlic, mushrooms, truffles, gherkins, radishes, mixed pickles, mustard, pepper, heavy and sweet wines, port, sherry, champagne, liqueur, chocolate, and strong coffee.

The chief dietary usually prescribed (with few variations) for gouty patients in Carlsbad is as follows:—Breakfast, weak tea (about 200 grammes), with cream (50 grammes), biscuits (60 to 100 grammes), butter (15 grammes), two soft-boiled eggs. Dinner, bouillon, either clear or with pearl-barley or rice (30 grammes), fish, either trout, or pike, or perch (100 grammes), with melted butter, lemon-juice and potatoes (50 grammes), roast veal (100 grammes), or mutton cutlets, or roast fowl, spinach, cheese (30 grammes), stewed fruit (100 grammes), bread (150 grammes). The beverages may consist of clear spring-water, or of one of the above-mentioned mineral waters, or half-pint of light Bordeaux with water, or two tablespoonfuls of whiskey with a half-bottle of mineral water. The same quantity and quality of drinks may be allowed at supper. Supper, either bouillon, or milk, or weak tea with milk, biscuits, lean bacon, or one or two eggs, and jam.

The dietary of patients suffering from gouty complications requires modification. Thus in diabetes the carbohydrates must be restricted, and fats and albuminoids permitted in larger amounts. In albuminuria, the nitrogenous elements must be reduced and rather more carbohydrates allowed. When obesity is present, the fats and carbohydrates must be diminished and more proteids given.

So far as clothing is concerned, great care is taken that the patients do not get cold or wet. Particular attention must be paid to the boots, which should be comfortable, so as not to interfere with the circulation.

In cases of advanced chronic gout, the more the internal organs are affected, the less must specific treatment be insisted on; and the patient must be treated on general principles. Apart from the special treatment of heart and kidneys, the constitution must be supported and all weakening influences avoided. If a definite cachetic condition has declared itself, all efforts directed to the treatment of gout proper must be abandoned, and care taken to keep up the strength of the body—quinine, iron, and arsenic being indicated. I have seen excellent results from the use of the water known as Levico, containing iron and arsenic, as also of Roncegno. If, as a result of stimulating treatment, the general state of the health is improved, then the ordinary dietetic and other treatment of gout may be resumed.

APPENDIX.

LITERATURE ON GOUT.

Bergman, Torbern: *Opuscula*, vol. iv., p. 387, quoted by Crell, *Die neuesten Entdeckungen in der Chemie*, 1776, Bd. ii., S. 232.

Butler: *A Treatise on the disease commonly called Angina pectoris*, 1791.

Caelius Aurelianus: *De Morbis acutis et chronicis.* Morb. chron., lib. v., cap. ii. Amsterdami, 1755.

Cullen: *Anfangsgründe der Arzneiwissenschaft.* Aus dem Englischen. Leipzig, 1778.

Demetrius Pepagomenus: *De podagra Liber*, quem ab eo petivit Imperator Michael Palaeologus. Paris, 1558.

Forbes: *A Treatise on the Gravel and Gout.* London, 1793.

De Haën: *Heilungsmethode.* Deutsch von Platzer. Liepzig, 1783, Band vi.

Lee, John: *A narrative of a singular gouty case.* London, 1782. Quoted by Legalchez.

Murray: *Ueber den gichtischen Tripper.* Aus dem Lateinischen. Göttingen, 1794.

Musgrave: *De Arthritide symptomatica.* London, 1703.

Musgrave: *De Arthritide anomala.* Oxford, 1707.

Pearson: "Experiments and Observations, tending to show the Composition and Properties of Urinary Concretions." Read 14 Dec., 1797. *Philosophical Transact. of the Royal Society, London*, 1798, p. 15.

Sauvages: *Nosologia methodica.* Amstelod., 1768.

Scheele, Karl Wilh.: *Examen chemicum calculi urinarii. Opuscula*, 1776, ii., p. 73.

Sydenham, Thomas: *Opuscula omnia. Tractatus de Podagra et Hydrope.* London, 1683.

Tennant: "Des nodosités des gouteux." *Journal de Physique*, 1797.

Watson: *Medical communications*, 1783.

Whyte, Robert: *Observations on the nature, causes, and cure of the disorders which have been called nervous.* London, 1765, p. 166.

Wollaston: "On Gouty and Urinary Concretions." *Philosoph. Transactions*, 1797, p. 386.

Barlow, Edward: *An Essay on the medical efficacy and employment of the Bath Waters*, 1822, pp. 113 and 114.

Barthez: *Traité des maladies goutteuses*, 1802. Deutsche Uebersetzung. Wien, 1807.

Beer, G. J.: *Lehre von den Augenkrankheiten*. Wien, 1817, Bd. ii., S. 544.

Bostock: "Case of a Periodical Affection of the Eyes and Chest." *Med.-chirurg. Transact.* London, 1819, vol. x.

Cadet de Vaux: *Neue Heilmethode der Gicht und des Rheumatismus*. Ilmenau, 1826.

Gendrin: *Leçons sur les maladies du cœur et des gros vaisseaux*, 1842, p. 625.

Graves: *Klinische Beobachtungen*. Deutsch von H. Bressler. Leipzig, 1843.

Guilbert: *De la goutte et les maladies goutteuses*. Paris, 1820.

Haygarth: *Clin. Hist. of diseases. Acute Rheumatism and nodosity of the joints*, 1805.

Heberden: *Opera medica*. Recogn. L. H. Friedländler. Leipzig, 1831. Cap. xxviii., p. 78.

Johnson, Th.: *Ueber die Natur der Gicht*. Deutsch. Halberstadt, 1820.

Kreysig: *Die Krankheiten des Herzens*. Berlin, 1815. Bd. i., S. 155; Bd. ii., S. 551.

Landré-Beauvais: *Doit on admettre une espèce de goutte asthénique ou primitive?* Paris, 1800.

Mazuyer: "De la présence de l'acide urique dans le sang considérée comme cause de la goutte." *Arch. gén. de Médic.* 1826, t. xi., p. 132.

Moore, James: "On Gouty Concretions or Chalk-stones." Read 18 March, 18(07). *Medico-chirurg. Transactions*, I., London, 1815.

Naumann: *Handbuch der medicinischen Klinik*. Berlin, 1834, Bd. iv., Abth. i., S. 83.

Parkinson: *Observations on the nature and cure of gout*. London, 1805.

Prout: *An inquiry into the Nature and Treatment of the Diabetes, Calculus, &c.* London, 1825.

Quarrier: *Edinburgh Med. and Surg. Journ*, 1808, vol. iv., p. 459.

Robertson: *The nature and treatment of Gout*. London, 1845.

Scudamore: *Natur und Heilung der Gicht*. Deutsch von Hesse. Halle, 1819.

Stosch: *Versuch einer Ontologie und Therapie des Diabetes mellitus*. Berlin, 1828.

Tanquerel des Planches: *Maladies de Plomb*. Paris, 1839.

Albers: *Die marasmische Knochengicht*. *Deutsche Klinik*, 1850, No. 25, S. 269.

Becquerel et Rodier: *Chimie phatologique*. Paris, 1854, p. 548.

Bence-Jones: *On gravel, calculus and gout*. London, 1842. Deutsch von Hoffmann. Braunschweig, 1845.

Bence-Jones: Remarks in the Discussion on Garrod's paper on "Chronic gout" at the Patholog. Society of London. *The Lancet*, 1856, vol. i., p. 98.

Bramson: "Arthritische Erkrankung der Gelenkknorpel." *Zeitschrift für rationelle Medicin*, 1845, Bd. iii., S. 175.

Castelnau: "Observations et réflexions sur la goutte et le rhumatisme." *Arch. génér. de médic.*, 1843, vol. iii., p. 285.

Critchett: *Med. Times and Gazette*, 1858, vol. i., p. 62.

Gairdner: *Die Gicht*. Deutsch von C. Braun. Wiesbaden, 1858.

Genth: *Untersuchungen über den Einfluss des Wassertrinkens auf den Stoffwechsel*. Wiesbaden, 1856.

Gintrac: *Cours de pathologie interne*, 1853, t. ii. and v.

Gueneau de Mussy: "Gravelle pharyngienne." *L'Union médicale*, 1856, No. 18, p. 76.

Laycock: *Treatise of the nervous system of women*, 1840, p. 163.

Ledwick, T. Hawkesworth: "Observations on the Pathological Relations of Local Gangrene to Constitutional Gout, &c." *Dublin Quarterly Journal of Medical Science*, 1858, vol. xxii., p. 40.

Liebig: *Jahresbericht über die Fortschritte der Chemie*, 1849, S. 531.

Marchand, R. F.: *Lehrbuch der physiologischen Chemie*. Berlin, 1844, S. 107.

Petit, Charles: "De l'emploi des eaux minérales de Vichy." *Journal de Pharmacie*, Oct. 1841.

Rayer: *Maladies des reins*. Paris, 1839.

Scott, Alison: "Arthritis occurring in the course of Paralysis." *Lancet*, 1847, i., p. 276.

Strahl und Lieberkühn: *Harnsäure im Blute und einige neue Bestandteile des Urins*. Berlin, 1848.

Todd: *Practical Remarks on Gout*. London, 1843.

Turck: *Traité de la goutte et des maladies goutteuses*. Paris, 1837.

Wöhler und Liebig: "Untersuchungen über die Natur der Harnsäure." *Annalen der Chemie und Pharmacie*, 1838, xxvi., S. 241.

Wöhler und Frerichs: "Ueber Veränderungen, welche namentlich organische Stoffe beim Uebergang in den Harn erfahren." *Annalen der Chemie und Pharmacie*, 1848, Bd. lxv., S. 335.

Ball: *Du rhumatisme viscéral. Thèse d'aggrégation*. Paris, 1866.

Barclay: *Gout and Rheumatism in relation to Disease of the Heart*. London, 1866.

Bartels: "Untersuchungen über die Ursachen einer gesteigerten Ausscheidung der Harnsäure in Krankheiten." *Deutsches Archiv für klinische Medicin*, 1866, Bd. i., S. 13.

Bazin: Article, "Arthritis," in *Dictionn. Encyclop. des Scienc. Médicales*, 1867, vol. vi., p. 359.

Begbie, James: *Contributions to Practical Medicine*. Edinburgh, 1862.

Bence-Jones: "On the Composition of the Amorphous Deposit of Urates in Healthy Urine." *Journal of the Chemical Society of London*, 1862, vol. xv., p. 201.

Berthier: "De la folie goutteuse." *Annales médico-psychologiques*. Paris, 1869, v. ser., t. i., p. 389.

Boose: *Ueber den Einfluss der Arzneimittel auf die Ausscheidung der Harnsäure. Inaugural-Dissertation*. Dorpat, 1862.

Braun: *Beiträge zu einer Monographie der Gicht*. Wiesbaden, 1860.

Brinton: "On Spasmodic Stricture of the Œsophagus." *Lancet*, 6 Jan., 1866, p. 1.

Budd: "Researches on Gout." *Medico-chirurgical Transact.*, 1855, p. 234.

Budd: *Krankheiten des Magens*, S. 93. Deutsch von Langenbeck. Göttingen, 1856.

Charcot et Cornil: "Contribution à l'étude des altérations anatomiques de la goutte et en particulier du rein chez les goutteux." *Mémoires de la Soc. de Biologie*, 1863, p. 153.

Corradi: "Della odierna diminuzione della podagra e delle sue cause." *Mem. dell' Acad. delle Scienze dell' Instituto di Bologna*, 1860, vol. x.

Dagonet: *Traité des maladies mentales*, 1862, p. 210.

Debout: "La goutte chez les enfants." *Union méd.*, 1869, p. 318.

Dickinson: *On the Pathology and Treatment of Albuminuria*. London, 1868.

Eckart, A.: *Die acute Gicht*. München, 1864.

Fenwick: *The Morbid States of the Stomach*. London, 1868, p. 55.

Galtier-Boissière: "De la goutte, de sa nature, de ses causes et de son traitement." *Thèse de Paris*, 1859.

Garrod, Alfred Baring: *Die Natur und Behandlung der Gicht*. Deutsch von Eisenmann. Würzburg, 1861.

Garrod: Article, "Gout," in *Reynold's System of Medicine*. London, 1866.

Garrod, Archibald E: "Goulstonian Lectures on Diabetes." *Brit. Med. Journ.*, 1857, p. 319.

Greenhow: "Clinical Lectures on Chronic Bronchitis." *Lancet*, 1867, vol. i., pp. 199 and 231.

Greenhow: "Clinical Lectures on Gouty Bronchitis." *Lancet*, 1867, vol. i., pp. 293 and 479.

Greenhow: *On Chronic Bronchitis as especially connected with Gout, Asthma, and Diseases of the Heart*. London, 1869.

Gubler: "De la tumeur dorsale des mains dans les paralysies saturnines des extenseurs des doigts." *L'Union médicale*, 1868, No. 78-80, p. 334.

Halma, Grand: *Etude clinique sur deux cas de goutte saturnine*, 1866.

Harvey, W.: *Rheumatism, Gout, and Neuralgia as affecting the Head and Ear, with Remarks on some forms of Headache in connection with Deafness*. London, 1852.

Hebra: *Hautkrankheiten*. *Virchow's Handbuch der speciellen Pathologie und Therapie*. Erlangen, 1860, Bd. iii.

Koch: "Ueber die Entstehung der Bersteinsäure im menschlichen Organismus." *Zeitschrift für rationelle Medicin*, 1865, iii. R., Bd. xxiv., S. 264.

Lanceraux: "Endocardite mitrale avec dépôts uratiques." *Gaz. médic. de Paris*, 1868, p. 187.

Legrand du Saulle: "Epilepsie goutteuse." *Gaz. des Hôpit.*, 1868, p. 506.

Lynch: "Some Remarks on the Metastasis of Diseases. Action to the Brain in Gout and other Diseases." *Dublin Quarterly Journal of Med. Science*, 1856, vol. xxi., p. 276.

Malherbe: *Des affections viscérales dans la goutte et le rhumatisme chroniques. Thèse de Paris*, 1866.

Martini e Ubaldini: "Recherches sur la composition de la sueur d'un goutteux." (From *Giornale veneto di scienze mediche*.) *Presse Médicale Belge*, 1866, No. 41.

Menjaud: *De la retraction spontanée et progressive des doigts dans ses rapports avec la goutte et le rhumatisme goutteux. Thèse de Paris*, 1861.

Naunyn: "Ueber die Chemie der Transsudate und des Eiters." *Reichert und Dubois' Archiv*, 1865.

Naunyn und Ries: "Ueber Harnsäureausscheidung." *Reichert-Dubois' Archiv*, 1869, S. 381.

Neubauer: "Ueber die Zersetzung der Harnsäure im Thierkörper." *Annalen der Chemie und Pharmacie*, 1856, Bd. i. c., S. 211.

Paget, James: "On Gouty and some other forms of Phlebitis." *St. Bartholomew's Hospit. Reports*, 1866, vol ii., p. 82.

Potton: *De la goutte et du danger des traitements empiriques*, 1860.

Ranke, H.: *Beobachtungen und Versuche über die Ausscheidung der Harnsäure beim Menschen*. München, 1858.

Ritter: "Zur Geschichte der Cynanche arthritica nebst Mittheilung der Beobachtung eines speciellen Falles derselben." *Württemberger ärztliches Correspondenzblatt*, 1863, No. 19.

Schroeder van der Kolk: "Uras calcis in de Rokken der Aderen bij Knobbeljicht." *Nederlandtsch Lancet*, iii. Ser., 1853, iii. Jahrgang, S. 97. (See *Cannstadt's Jahresberichte*, 1853, Bd. ii., S. 47.)

Seegen: "Beitrag zur Casuistik der Melliturie." *Virchow's Archiv*, 1864.

Seegen: "Physiologisch-chemische Untersuchungen über den Einfluss des Karlsbader Mineralwassers auf einige Factoren des Stoffwechsels." *Wiener medicinische Wochenschrift*, 1860, No. 21.

Stokes, William: *Die Krankheiten des Herzens und der Aorta.* Deutsch von Lindwurm. Würzburg, 1855, S. 429.

Stokvis: "Beiträge zur Physiologie der Harnsäure." *Archiv für holländische Beiträge*, ii. Serie, 1860, S. 260. See *Schmidt's Jahrbücher*, 1861, Bd. cix., S. 4.

Strecker: "Bildung von Glykokoll aus Harnsäure." *Annalen der Chemie und Pharmacie*, 1868, Bd. cxlvi., S. 142.

Todd: *Clinical Lectures on certain Diseases of Urinary Organs*, 1859.

Toynbee: *Diseases of the Ear*, 1860. Deutsch von Moos. Würzburg, 1863.

Trousseau, A.: *Medicinische Klinik des Hôtel-Dieu.* Deutsch von Gulmann. Würzburg, 1867, Bd. ii., S. 429, und Bd. iii.

Virchow: "Seltene Gichtablagerungen." *Virchow's Archiv*, 1868, Bd. xliv., S. 137.

Voit und Hoffmann: "Ueber das Zustandekommen der Harnsäuresedimente." *Sitzungsberichte der königlich baierischen Akademie der Wissenschaften*, 1868, ii., S. 104.

Wells, Spencer: *Practical Observations on Gout and its Complications.* London, 1854.

Zaleski: *Untersuchungen über den urämischen Process und die Function der Nieren.* Tübingen, 1865.

Adams: *Observations on Contraction of Fingers, commonly called Dupuytren's Contraction*, 1873.

Alexander von Tralles: Ausgabe von Puschmann. Wien, 1879, Bd. ii., S. 500, 12. Buch: "Ueber das Podagra."

Bartels: *Nierenkrankheiten.* Leipzig, 1875, S. 375.

Beneke: *Grundlinien der Pathologie des Stoffwechsels*, 1874.

Bouchard: *Comptes rendus de la Soc. de Biologie*, 1873.

Bouilly: "Altérations articulaires et viscérales diverses observées chez un goutteux." *Bull. de la Soc. anatom.*, 1872, vol. xlvii., p. 275.

Bourneville: *Etudes cliniques et thermométriques sur les maladies du système nerveux.* Paris, 1872, p. 58.

Braun, Jul.: *Klinische und anatomische Beiträge zur Kenntniss der Spondylitis deformans.* Hanover, 1875.

Brogniart: *Contribution à l'étude de la goutte viscérale.* Paris, 1875.

Brogniart: *Contribution à l'histoire du diabète goutteux.* Paris, 1876.

Budin: "Goutte datant de vingt ans; usage immodéré de la liqueur Laville." *Bull. de la Soc. anat.*, 1873, p. 709.

Buhl: *Mittheilungen aus dem pathologischen Institut zu München*, S. 50. Stuttgart, 1876.

Charcot: *Leçons cliniques sur les maladies des vieillards et les maladies chroniques.* Paris, 1874.

Charreyron: *Des manifestations de la goutte sur les muqueuses. Thèse de Paris*, 1876.

Collin: *Du diognostic de la congestion pulmonaire de nature arthritique et de son traitement*, 1877.

Cornillon: *Rapports du diabète avec l'Arthritis.* Paris, 1878.

Coupland, Sydney: "Gouty deposits on the aortic valves." *Transact of the Pathol. Soc. of London*, t. 1873, vol. xxiv., p. 69; *Lancet*, 1873, vol. i., p. 447.

Darney: *Contribution à l'étude de la goutte. Thèse*, 1874.

Duckworth: "Some irregular Manifestations of Gout." *St. Barthol. Hosp Rep.*, 1879, vol. xv.

Durand: *Étude sur la goutte saturnine. Thèse de Paris*, 1878.

Féréol: "Des nodosités cutanées éphémères chez les arthritiques." *Congrès de Paris*, 1879.

Gueneau de Mussy: *Clinique médicale*, 1875, t. i.

Hagen: Ueber einen Fall von Lähmung der Schultermusculatur und des Musc. serratus ant. maior nach acutem Gelenkrheumatismus." *Münchener medicinische Wochenschrift*, 1879, Bd. xxxvi., No. 25 and 26.

Heidenhain: "Versuche über den Vorgang der Harnabsonderung." *Pflüger's Archiv*, 1874, Bd. ix., S. 23.

Hood, Peter: *A treatise on gout, rheumatism, and allied affections*. London, 1871.

Hutchinson: "On the hereditary transmission of gout." *Medic. Times and Gaz.*, 20 May 1876.

Jaccoud et Labadie-Lagrave: Art. "Goutte" in *Dictionn. de Méd. et Chir. pratiques*, par Jaccoud. Paris, 1873, t. xvi.

Lancereaux: *Atlas d'anatomie pathologique*. Paris, 1871, p. 214, pl. 22, fig. 4; p. 496, pl. 54, fig. 8.

Lancereaux: Article "Rein" in *Dictionn. Encycl. des Sciences Médic.*, par Déchambre. Paris, 1876, 3 s., t. iii.

Lancereaux: *Traité d'anatomie pathologique*. Paris, 1879 bis 1881, t. ii., p. 729.

Leber: "Beiträge zur Ætiologie innerlicher Augenentzündungen." *XII. Versammlung der ophthalmologischen Gesellschaft zu Heidelberg*, 1879, S. 133.

Lecorché: *Traité de la goutte*. Paris, 1884.

Lecorché: *Traité du diabète*. Paris, 1877, p. 273.

Litten: "Ein Fall von schwerer Gicht mit Amyloiddegeneration." *Virchow's Archiv*, 1876, Bd. lxvi., S. 129.

Liveing: *On Megrim, sick-headache, and some allied disorders*. London, 1873.

Milton: *Edinburgh Med. Journ.*, Dec., 1876 (*Vierteljahresschrift für Dermatologie*, 1877, S. 585).

Mounier-Lompré: *De la goutte dans ses rapports avec les lésions traumatiques. Thèse de Paris*, 1876.

Moxon: "Case of Gout of the Stomach and Phlegmonous Colitis." *Transact. of the Path. Soc. of London*, 1871, xxii., p. 139.

Murchison: *Clinical lectures on diseases of the liver*. London, 1877.

Ollivier: "Contribution à l'histoire de la goutte spinale." *Archives de Physiologie*, 1878, vol. v., p. 455.

Paget: *British Med. Journal*, 1875, i., p. 704.

Paget: *Clinical Lectures and Essays*, 1877, p. 292.

Pawlinoff: "Die Bildungsstätte der Harnsäure im Organismus." *Virchow's Archiv*, 1875, Bd. lxii., S. 57.

Pouget: *Étude de la goutte. Thèse de Paris*, 1878.

Rees Owen: *Brit. Med Journ.*, 1877, i., p. 225.

Renaut: *L'intoxication saturnine chronique*, 1875.

Reynolds I. Russell: "Some affections of the nervous system dependent upon a gouty habit." *Brit. Med. Journ.*, 1877, ii., p. 842.

Salkowski, E.: "Bildung von Allantoin aus Harnsäure im Thierkörper." *Berichte der deutschen chemischen Gesellschaft*, 1876, Bd. ix., S. 719, and 1878, Bd. xi., S. 500.

v. Schroeder, Waldemar: "Ueber die Verwandlung des Ammoniaks in Harnsäure im Organismus der Hühner." *Zeitschrift für physiologische Chemie*, 1878, Bd. ii., S. 228.

Sée, G.: "Etude sur l'acide salicylique et les salicylates." *Bull. de l'Acad. de Médec.*, 1877.

Senator: "Gicht," in *v. Ziemssen's Handbuch der speciellen Pathologie, und Therapie*, 2. Aufl., 1879, Bd. xiii., 1, S. 133.

Thompson: *Die chirurgischen Krankheiten der Harnwege.* Deutsche Uebersetzung. Berlin, 1877, S. 209.

Tuckwell: "On clotting of the blood in gout and chlorosis." *St. Bartholom. Hosp. Reports*, 1874, vol. x., p. 23.

Abeles, M.: "Ueber Harnsäure im Blute und einigen Geweben und Organen." *Wiener medicinische Jahrbücher*, 1877, Bd. lxxxiii., S. 479.

Arndt, R.: *Die Neurasthenie.* Wien, 1885, S. 123.

Axenfeld et Huchard: *Traité des névroses.* Paris, 1888.

Belliard: *Des manifestations cérébrales de la goutte. Thèse de Paris*, 1882.

Boucheron: "De la présence anormale de l'acide urique dans les secrétions salivaire, gastrique, nasale, pharyngée, sudorale, utérine, et dans le sang menstruel." *Acad. des Sciences*, Août, 1881.

Bouloumié: "Atrophies goutteuses et leur traitement." *Bull. de la Soc. de Therapeut.*, 1881, No. 11.

Bourdillon: *Psoriasis et arthropathies. Thèse de Paris*, 1888.

Brissaud: *Recherches anatomo-pathologiques et physiolog. sur la contracture permanente des hemiplégiques.* Paris, 1880.

Bum, Anton: "Ueber den Einfluss der Massage auf die Harnsecretion." *Zeitschrift für klinische Medicin*, 1888, Bd. xv., S. 248.

Burchard: *Ueber den Einfluss des Kohlensauren, respective citronensauren Natrons auf den Stoffwechsel. Inaugural-Dissertation.* Dorpat, 1889.

Buzzard: "On some forms of paralysis from peripheral neuritis." *Harveian Lectures for* 1885. London, 1886.

Camerer: *Zuckerharnruhr, Corpulenz, Gicht und chronische Nierenkrankheiten.* Tübingen, 1888.

Cantani: *Specielle Pathologie und Therapie der Stoffwechselkrankheiten.* Deutsch von Hahn. Berlin, 1880, Bd. ii.

Charcot: *Leçons sur les maladies du foie et des reins.* Paris, 1888.

Charcot: *Leçons sur les maladies du système nerveux*, t. 1880, vol. i.

Charcot: "Sur l'atrophie qui succède à certains lésions articulaires." *Progrès Médic.*, 1882, Nos. 20, 21.

Chittenden: "Ueber den Einfluss von Urethan, Antipyrin und Antifebrin auf den Eiweissumsatz." *Zeitschrift für Biologie*, 1889, Bd. xxv., S. 496.

Clark, Andrew: "Report on a case of gouty melancholia." *Journal of Mental Sciences*, Oct. 1880, p. 343.

Colasanti: *Richerche sperimentali sulla formazione dell'acido urico.* Roma, 1881. Deutsch in Moleschott's *Untersuchungen zur Naturlehre*, Bd. xiii.

Coriveaud: "De l'antagonisme de la phthisie pulmonaire et de la goutte." *Bordeaux Médical*, 1887, pp. 260, 267.

Cornil et Ranvier: *Manuel d'histologie pathologique.* 2. édit., Paris, 1881 bis 1884, i., p. 476, ii., p. 603.

Cornillon: "Amyotrophie consécutive à deux accès de goutte, simulant l'atrophie musculaire progressive." *Progrès médical*, 1883, p. 405.

Corlett: See *Monatshefte für Dermatologie*, 1887, Bd. vi., S. 622.

Da Costa: "The nervous symptoms of lithæmia." *Amer. Journ. of Med. Sciences*, Oct. 1881.

Debout d'Estrées: "Contribution à l'histoire de la goutte anormale." *Gaz. des Hôp.*, 1882, No. 77.

Debout d'Estrées: "Orchite goutteuse." *Gaz. des Hôp.*, 1885, p. 84.

Debout d'Estrées: "Goutte des glandes." *Acad. de Méd. de Paris*, 12 Mai, 1885.

Debout d'Estrées: "Some new facts concerning gouty parotitis and gouty orchitis." *Medic.-chirurg. Transact., London*, 1887, p. 217.

Duckworth: "On gout considered as a trophoneurosis." *Brit. Med. Journ.*, 1881, vol. i., p. 463.

Durand-Fartel: "Considérations sur la théorie de la goutte." *Union médicale*, 8–10 Juillet, 1883.

Ebstein, W.: "Beiträge zur Lehre von der Gicht." *Deutsches Archiv für klinische Medicin*, 1880, Bd. xxvii., S. 1.

Ebstein: "Ueber den gichtischen Process." *Verhandlungen des zweiten medicinischen Congresses*, 1882.

Ebstein: *Die Natur und Behandlung der Gicht.* Wiesbaden, 1882.

Ebstein: *Die Natur und Behandlung der Harnsteine.* Wiesbaden, 1884.

Ebstein: *Das Regimen bei der Gicht.* Wiesbaden, 1885.

Ebstein: "Die Natur und Behandlung der Gicht." *Referat auf dem achten Congress für innere Medicin.* Wiesbaden, 1889.

Ebstein: *Beiträge zur Lehre von der harnsauren Diathese.* Mit Dr. Oelker und Dr. Sprague. Wiesbaden, 1891.

Ebstein: "Angina pectoris neben Arthritis uratica und Diabetes mellitus," *Berliner klinische Wochenschrift*, 1895, Nos. 23–25, S. 493, 522, 543.

Ebstein: "Ueber die Beziehungen der sogenannten harnsauren Diathese zur Leukämie." *Virchow's Archiv*, 1898, Bd. cliv., S. 349.

Ebstein: "Zur Lehre von der gichtischen Neuritis." *Deutsche medicinische Wochenschrift*, 1898, No. 31, S. 489.

Ebstein: "Ueber die Stellung der Fettleibigkeit, der Gicht und der Zuckerkrankheit im nosologischen System." *Verhandlungen der VII. Naturforscherversammlung in Düsseldorf*; Abtheilung für innere Medicin. *Deutsche medicinische Wochenschrift*, 1898, No. 44, S. 693.

Fothergill, J. M.: "Gouty neuroses of the Heart." *Edinburgh Medical Journal*, November 1884, p. 393.

Gailhard: "Prostatite avec abcès chez un goutteux." *Bull. de la Soc. médic. des Hôp.*, 1885; *Gaz. des Hôp.*, 1885, p. 493.

Galezowski: "La goutte et les affections oculaires d'origine goutteuse." *Journal de therapeutique de Gubler*, 1882.

Goudot: *Etude sur la goutte saturnine.* Paris, 1883.

Guyot: "Orchite goutteuse alternant avec des manifestations articulaires." *Gaz. des Hôpit.*, 1885, p. 36.

Guyot: *L'arthritis, maladie constitutionnelle.* Paris, 1890.

Haig: "On the influence of diet on headache." *The Practitioner*, 1886, t. xxxvi., p. 179.

Harrison, Reginald: "Prostatic Gout." *Lancet*, 1883, vol. ii., p. 890.

Hermann, Aug.: "Ueber die Abhängigkeit der Harnsäureausscheidung von Nahrungs- und Genussmitteln mit Rücksicht auf die Gicht." *Deutsches Archiv für klinische Medicin*, 1888, Bd. xliii.

Huchard: "Des congestions pulmonaires chez les arthritiques." *Congrès pour l'avancement des sciences*, 1883.

Hutchinson: "Lecture introductory to the Study of the Arthritic Diathesis." *Med. Times and Gaz.*, 1 Jan. 1881.

Hutchinson: "On the relation of certain diseases of the eye to gout." *Brit. Med. Journ.*, 1884, vol. ii., p. 995.

Jacob: *Ueber die Bleikrankheiten im Oberharz und deren Beziehungen zu Gicht und Schrumpfniere.* Inaugural-Dissertation, Göttingen.

Jacobi: "Pharmakologische Untersuchungen über das Colchicumgift." *Archiv für experimentelle Pathologie und Pharmakologie*, Bd. xxvii.

Jollye: "A case of gouty peripheral neuritis." *Bristol Medico-chirurgical Journal*, 1886, vol. ii., p. 28.

Kusmanoff: *Ueber die Ausscheidung der Harnsäure bei absoluter Milchdiät.* Inaugural-Dissertation, Dorpat, 1885.

Latil: "Orchite survenant brusquement sept jours après un accès de goutte articulaire." *Bull. de la soc. méd. des Hôpit.*, 1885, No. 3.

Legalchez-Baron: *Des manifestations de la goutte sur les organes génitaux. Thèse de Paris*, 1886.

London, B.: "Ueber den Einfluss des Kochsalz- und Glaubersalzhaltigen 'Mineralwassers' auf einige Factoren des Stoffwechsels." *Zeitschrift für klinische Medicin*, Band xiii., H. 1, 1884.

Lorimer: "Saturnine gout and its distinguishing marks." *Brit. Medic. Journ.*, 24 July, 1886.

Meldon: "Pathology and treatment of gout." *Brit. Med. Journ.*, 1881, i., p. 466.

Moebius: "Ueber Migräne." *Centralblatt für Nervenheilkunde*, 1885, viii., S. 244.

Moore, Norman: "Specimens from a case of gout." *Transactions of the Pathological Society of London*, 1882, p. 271.

Moore: "Some observations on the morbid anatomy of gout." *St. Bartholomew's Hosp. Reports*, 1887, vol. xxiii., p. 289.

Myrtle: "Eczema and Psoriasis." *Lancet*, 1887.

Ord: "On some disorders of Nutrition related with affections of the nervous system." *Brit. Med. Journ.*, 1884, ii., p. 205.

Pechaud: "Orchite du testicule gauche disparaissant pour être remplacée par un accès de goutte." *Bull. de la Soc. Méd. des Hopitaux*, 1885.

Pedell: "Ueber Gicht und Nierenschrumpfung bei Bleiintoxication." Berliner Verein für innere Medicin, 1884. *Deutsche medicinische Wochenschrift*, 28 Februar, 1884.

Pfeiffer, Emil: "Zur Aetiologie und Therapie der Gicht." *Verhandlungen des V. Congresses für innere Medicin, Wiesbaden*, 1886, S. 444.

Pfeiffer: "Die Natur und Behandlung der Gicht." *Verhandlungen des VIII. Congresses für innere Medicin*, 1889, S. 166.

Quincke: "Ueber acutes umschriebenes Hautödem." *Monatshefte für praktische Dermatologie*, 1882, i., S. 129.

Roose, Robson: *Die Gicht und ihre Beziehungen zu den Krankheiten der Leber und der Nieren.* Uebersetzt nach der 4. Aufl. von Krakauer. Wien und Leipzig, 1887.

Salkowski: "Ueber die Grösse der Harnsäureausscheidung und den Einfluss der Alkalien auf dieselbe." *Virchow's Archiv*, 1889, Bd. cxvii., S. 570.

Saundby: "Gout and Rheumatism." *Med. Times and Gaz.*, 8 Jan. 1881.

Schindler: "Beitrag zur Kenntniss des Adenins, Guanins und ihrer Derivate." *Zeitschrift für physiologische Chemie*, 1889, xiii., S. 432.

Tichborne: "On the elimination of uric acid by the skin." *Brit. Med. Journ.*, 1887, ii., p. 1097.

Virchow: "Ueber Nephritis arthritica." *Berliner klinische Wochenschrift*, 1884, xxi., No. 1.

Armstrong: "The value of an exclusive red meat diet in certain cases of chronic gout." *Lancet*, 3 July, 1897.

Aronsohn, Ed.: "Zur Natur und Behandlung der Gicht und über die Bedeutung der Emser Wilhelmsquelle." *Deutsche medicinische Wochenschrift*, 1890, No. 18.

Ascoli, G.: "Ueber die Stellung der Leber im Nucleïnstoffwechsel." *Pflüger's Archiv*, 1898, Bd. lxxii., S. 340.

Baccelli: "Patogenesi della gotta." *Gaz. degli Ospedali*, 1898, vol. xix., p. 55.

Badt: " Ueber Harnsäureausscheidung im Urin während des acuten Gichtanfalles." *Zeitschrift für klinische Medicin*, 1899, Bd. xxxvii., S. 546.

Bäumler: " Referat über den chronischen Gelenkrheumatismus." *Verhandlungen des* 15. *Congresses für innere Medicin zu Berlin.* Wiesbaden, 1897.

Bain: " The actions of various drugs and diets on the excretion of nitrogen in gout." *Brit. Med. Journ.*, April, 1900.

Barjou: *Du Syndrome rhumatismal chronique déformant. Étude radiographique des arthropathies déformantes. Thèse de Lyon*, 1897.

Baum: " Gout as a cause of ear disease." *Philad. Policl.* 1897, vol. vi., p. 306.

Baumgarten, Oswald: " Beitrag zur Aetiologie der primären Schrumpfniere." *Münchner medicinische Wochenschrift*, 1901, No. 1, S. 19.

Bendix, Ernst: "Wirkt die Harnsäure antiseptisch?" *Zeitschrift für klinische Medicin*, 1902, Bd. xliv., S. 165.

Benjamin: " Ueber Blei in der Leber eines Gichtkranken." *Charité-Annalen*, 1898, Bd. xxiii., S. 311.

Bennecke, Erich: " Beiträge zur Anatomie der Gicht." *Archiv für klinische Chirurgie*, 1902, Bd. lxvi., S. 658.

Bergmeister: " Ueber das Vorkommen von Störungen des Sehorganes bei gewissen Stoffwechselanomalien, insbesondere bei harnsaurer Diathese." *Wiener medicinische Wochenschrift*, 1894, No. 42 und 43.

Biesenthal,: " Ueber den Einfluss des Piperazin auf die harnsaure Diathese." *Archiv für pathologische Anatomie*, 1894, Bd. cxxxvii.

Bishop: "A new and successful treatment of hay-fever." *Americ. Med. News*, 1895, No. 8.

Blumenthal: " Ueber die Ausscheidung der Harnsäure nach Darreichung von Chinasäure." *Charité-Annalen*, 1900, Bd. xxv., S. 34.

Blumenthal und Lewin: " Ueber Sidonal." *Therapie der Gegenwart*, 1900, No. 4.

Bohland: " Ueber den Einfluss einiger Arzneimittel auf die Bildung und Ausscheidung der Harnsäure." *Münchener medicinische Wochenschrift*, 1899, No. 16, S. 505.

Bondzynski und Gottlieb: " Ueber Methylxanthin, ein Stoffwechselproduct des Theobromin und Kaffeïn." *Archiv für experimentelle Pathologie und Pharmakologie*, 1895, Bd. xxxvi., S. 45.

Bouchard: *Leçons sur les maladies par ralentissement de la nutrition.* 3rd Edition. Paris, 1890.

Brandenburg, Kurt: *Deutsches Archiv für klinische Medicin.* Bd. lviii., S. 82.

Brocq: " Le régime alimentaire dans les dermatoses." Festschrift für G. Lewin. *Zeitschrift für Dermatologie und Syphilis.* Berlin, 1895.

Brooke: " On the Pathology and Treatment of Pruritus." *British Medical Association in London*, 1895, see: *Archiv für Dermatologie*, 1897, Bd. xxxviii., S. 105.

Bruce, J. Mitchell: " The gouty heart." *The Practitioner*, Jan. 1895.

Buck, Albert H.: " Goutiness in its relations to diseases of the ear." *Transact. of the Americ. Otolog. Soc.* 1897, vol. vi., p. 439.

Bulkley, Duncan: " Clinical notes on Psoriasis." *Transactions of the Medical Society of the State of New York*, 1895.

Burian und Schur: " Ueber Nucleïnbildung im Säugethierorganismus." *Zeitschrift für physiologische Chemie*, 1897, Bd. xxiii., S. 55.

Buzzard: " On Vertigo of bulbar origin." *Lancet*, 1890, vol. i., p. 179.

Camerer: " Zur Lehre von der Harnsäure und Gicht." *Deutsche medicinische Wochenschrift*, 1891, Bd. xvii., S. 356.

Camerer: " Harnsäure, Xanthinbasen und Phosphorsäure im menschlichen Urin." *Zeitschrift für Biologie*, 1896, Bd. xxxiii., S. 139.

Camerer: "Beitrag zur Erforschung der stickstoffhaltigen Bestandtheile des menschlichen Urins, insbesondere der sogenannten Alloxurkörper." *Zeitschrift für Biologie*, 1897, Bd. xxxv., S. 206.

Camerer: *Der Gehalt des menschlichen Urins an stickstoffhaltigen Körpern.* Tübingen, 1901.

De la Camp: "Chinasäure und Gicht." *Münchener medicinische Wochenschrift*, 1901, No. 30, S. 1203.

Caro: "Ueber die Beziehungen epileptischer Anfälle zur Harnsäureausscheidung." *Deutsche medicinische Wochenschrift*, 1900, No. 19.

Ten Cate: *Beiträge zur gichtischen Diathese.* Inaugural-Dissertation. Göttingen, 1899.

Chittenden: "The influence of Alcohol on Proteïn Metabolism." *Journal of Physiology*, 1891, vol. xii., p. 220.

Claus: "Arthritisme, migraines et salophène." *Flandre médic.*, 1895, vol. ii., p. 43. Uebersetzt in: *Therapeutische Monatshefte*, 1895, Bd. ix., S. 598.

Cohn, Theodor: "Beitrag zur Kenntniss des Stoffwechsels nach Thymusnahrung." *Zeitschrift für physiologische Chemie*, 1898, Bd. xxv., S. 507.

Cornillon: "Influence du traumatisme sur l'apparition de la goutte." *Progrès médical*, 1897, p. 1.

Cousin: *De quelques symptomes communs au rhumatisme chronique et aux affections nerveuses.* Paris, 1890.

Danziger: "Ueber Angina urica." *Monatshefte für Ohrenheilkunde*, 1895, Bd. xxix., S. 14.

Dapper, Karl: "Ueber Harnsäureausscheidung beim gesunden Menschen unter verschiedenen Ernährungsverhältnissen." *Berliner klinische Wochenschrift*, 1893, Bd. xxx., S. 619.—*v. Noorden's Beiträge zur Lehre vom Stoffwechsel*, 1894, Heft 2.

Dapper: "Ueber den Einfluss der Kochsalzquellen auf den Stoffwechsel beim Menschen und die sogenannte 'curgemässe' Diät." *Zeitschrift für klinische Medicin*, 1896, Bd. xxx.

Debout d'Estrées: "Quelques formes rares de la goutte. Goutte de poumon, de l'œil, de l'oreille, des glandes sublinguales, de la parotide et du testicule." *Annales d'Hydrologie*, 1891, t. xxxvi.

Delpeuch: *Histoire des maladies. La goutte et le rhumatisme.* Paris, 1900.

Desnos: "Des hématuries chez les goutteux." *Annal. des malad. des organ. génito-urin.* 1899, vol. xvii.

Determeyer und Büttner: "Zur Therapie der harnsauren Diathese." *Deutsche medicinische Wochenschrift*, 1901, No. 21.

Drummond: "Note on 'neurotic' symptoms of uric-acidæmia in the young." *Lancet*, 1897, vol. i., p. 1338.

Duckworth: "On mental disorders dependent on toxæmia." *Lancet*, 24 November, 1900.

Duckworth: "The pathogeny of gout." *Lancet*, 25 August, 1900.

Duckworth: "Rheumatoid arthritis, Heberden's nodes, Dupuytren's contraction and some other arthritic conditions." *St. Bartholomew's Hosp. Rep.* 1900, vol. xxxv.

Dunin und Nowaczek: "Ueber Harnsäureausscheidung bei croupöser Pneumonie." *Zeitschrift für klinische Medicin*, 1897, Bd. xxxii., S. 1.

Ebstein: "Gicht." In Ebstein's und Schwalbe's *Handbuch der practischen Medicin*, Bd. iii., Theil 2, Stuttgart, 1901.

Ebstein: "Gicht." In v. Leyden's und Klemperer's *Deutsche Klinik*, 1902, Bd. iii., S. 98.

Ebstein und Nicolaier: "Ueber die Ausscheidung der Harnsäure durch die Nieren." *Virchow's Archiv*, 1896, Bd. cxliii., S. 337.

Ebstein und Sprague: "Notiz, betreffend die therapeutische Anwendung des Piperazin." *Berliner klinische Wochenschrift*, 1891, No. 14.

Eitner: *Ueber die Beeinflussung der Harnsäure und Alloxurbasen durch die Extractivstoffe des Fleisches.* Inaugural-Dissertation. Leipzig, 1896.

Elgood: "Note on a fatal case of lithæmic poisoning." *Lancet*, 18 February 1899.

Ewart: *Gout and Goutiness.* London, 1896.

Féré: "Accès périodiques de perversion instinctive chez un goutteux." *La Flandre méd.*, Juillet, 1894.

Fischer, Emil: "Synthesen in der Puringruppe." *Berichte der Deutschen chemischen Gesellschaft*, 1899, Bd. xxxii., S. 435.

Förster: *Versuche über die Beeinflussung der Harnsäureausscheidung mit specieller Berücksichtigung der Chinasäure und der chinasauren Salze* Inaugural-Dissertation Breslau, 1900.

Freudweiler: "Experimentelle Untersuchungen über die Entstehung der Gichtknoten." Züricher Habilitationsschrift, 1900. *Deutsches Archiv für klinische Medicin*, 1900, Bd. lxix., S. 155.

Friedländer: "Physikalische Therapie der gichtischen Gelenkerkrankungen." Goldscheider und Jakob's *Handbuch der physikalischen Therapie*, 1902, Theil II., Bd. 1, S. 96.

Fürst, L.: "Die Rolle der Alkalien, speciell des Kalkes in der anti-uratischen Behandlung." *Deutsche Medicinal-Zeitung*, 1897, No. 20, 21.

Garrod: "The use of Guaiacum in the treatment of chronic gouty affections and its value in warding off acute attacks." *Med. and Chirurg. Society*, 26 May, 1896. *Lancet*, 1896.

Garrod, Archibald E.: *A Treatise on Rheumatism and Rheumatoid Arthritis.* London, 1890.

Gellé: "Contribution à l'étude des affections auriculaires chez les goutteux ou les individus de race goutteuse." *Archiv. internat. d'Otol.*, September-October 1897, see: *Virchow-Hirsch's Jahresbericht*, 1897, Bd. ii., p. 394.

Gemmel: *Die Gicht.* Berlin, 1901.

Gerhardt, C.: "Uber das Verhältniss von Schuppenflechte zu Gelenkerkrankungen." *Berliner klinische Wochenschrift*, 1894, Bd. xxxi., S. 38.

Gerhardt, C.: "Ueber das Fieber der Gichtkranken." Gesellschaft der Charité-Aerzte in Berlin, 9 November, 1899. *Deutsche medicinische Wochenschrift*, Vereins beilage, 1899, No. 49, S. 300.

Glaser: "Ueber den Einfluss alkoholischer Getränke auf das Harnsediment des normalen Menschen." *Deutsche medicinische Wochenschrift*, 1891, No. 43.

Goodbody, Woodcock: "The action of Lysidin and Piperazin as uric acid solvents." *Brit. Medic. Journ.*, 3 October, 1896.

Goto, M.: "Ueber die Lösung der Harnsäure durch Nucleïnsäure und Thyminsäure." *Zeitschrift für physiologische Chemie*, 1900, Bd. xxx., S. 473.

Gowers: *Handbuch der Nervenkrankheiten.* Deutsch von Grube. Bonn, 1892. S. 63, 101, 105, 114, 314, 601.

Grawitz, E.: "Beobachtungen über ein neues harnsäurelösendes Mittel (Lysidin)." *Deutsche medicinische Wochenschrift*, 1894, No. 14.

Grosz: "Ueber Beziehungen einiger Dermatosen zum Gesammtorganismus." *Wiener klinische Wochenschrift*, 1899, No. 9.

Grube, Karl: "Ueber Psoriasis in Zusammenhang mit Gicht und Diabetes." *Berliner klinische Wochenschrift*, 1897, Bd. xxxiv., S. 1134.

Grube, Karl: "Ueber gichtische periphere Neuritis." *Münchener medicinische Wochenschrift*, 1899, No. 23.

Grube: "Ueber gichtische Affectionen des Herzens und gichtische periphere

Neuritis." Vortrag auf dem XX. Deutschen balneologischen Congress. Berlin, 1899. *Wiener medicinische Wochenschrift*, 1899, No. 25, S. 1191.

Grube, Karl: "Ueber gichtische Erkrankungen des Magens und Darms." *Verhandlungen des XVIII. Congresses für innere Medicin.* Wiesbaden, 1900.

Grube, Karl: "Ueber den Einfluss der Mineralwässer auf das Blut." *Zeitschrift für diätetische und physikalische Therapie*, 1902, Bd. vi., S. 334.

Haeser: *Der Einfluss des Alkohols auf die Harnsäureausscheidung.* Inaugural Dissertation. Greifswald, 1901.

Haig: *Uric acid as a factor in the causation of disease.* London, 1896. Deutsch von Birchner-Benner, Berlin, 1901.

Haig: "Variations in the excretion of uric acid produced by administration of acids and alkalies." *Journal of Physiology*, 1895, vol. viii.

Haig: "The effect of muscular work upon the alkalinity of the blood." *Lancet*, 1896, vol. i., p. 1095.

Haig: "The alkalinity of the blood in gout." *Brit. Med. Journ.*, 1898, pp. 1171 and 1294.

Haig and Oxon: "Further observations on the excretion of uric acid in epilepsy and the effects of drugs on the fits." *Brain*, 1896, vol. xix., p. 68.

Hallmeyer: *Die Gicht in ihren Beziehungen zu den Krankheiten des Ohres.* Inaugural-Dissertation. München, 1901.

Harley, George: "Gout in relation to liver disease." *Lancet*, 4 September, 1896.

Harley: "Zucker und Champagner bei Gichtkranken." *Lancet*, 1896.

Harris: "Renal hæmaturia without known lesions." *Philadelphia Med. Journ.*, 19 March, 1898.

Hedges: "The relation of Gout and Rheumatism to Dupuytren's contraction of palmar fascia." *St. Bartholom. Hospit. Reports*, 1897, t. xxxii., p. 119.

Herlant: "Untersuchungen über die Nucleïnsäure." *Archiv für experimentelle Pathologie und Pharmakologie*, 1900, Bd. xliv.

Hess und Schmoll: "Ueber die Beziehungen der Eiweiss- und Paranucleïnsubstanzen der Nahrung zur Alloxurkörperausscheidung im Harn." *Archiv für experimentelle Pathologie und Pharmakologie*, 1896, Bd. xxxvii., S. 243.

His, W., jun.: "Die Ausscheidung von Harnsäure im Urin der Gichtkranken, mit besonderer Berücksichtigung der Anfallszeiten und bestimmter Behandlungsmethoden." *Archiv für klinische Medicin*, Bd. lxv., S. 156.

His, W., jun.: "Experimentelle Untersuchungen über das Wesen der Gichtknoten." *Deutsches Archiv für klinische Medicin*, 1900, Bd. lxv., S. 618.

His, W., jun.: "Die Harnsäureablagerungen des Körpers und die Mittel zu ihrer Lösung." Vortrag auf der Versammlung Deutscher Naturforscher und Aerzte in Hamburg. *Therapie der Gegenwart*, Neue Folge, 1901, Bd. iii., S. 434.

His und Hagen: "Kritische Untersuchungen über den Nachweis von Harnsäure und Purinbasen im Blut und in thierischen Organen." *Zeitschrift für physiologische Chemie*, 1900, Bd. xxx., S. 350.

Hoffa: "Ueber die arthritische Muskelatrophie." XXI. Congress der Deutschen Gesellschaft für Chirurgie. *Deutsche medicinische Wochenschrift*, 1892, S. 754.

Hopkins and Hope: "On the relation of uric acid excretion to diet." *Journal of Physiology*, 1898, vol. xxiii., p. 271.

Horbaczewski: "Synthese der Harnsäure," *Monatshefte für Chemie*, 1892, Bd. iii., S. 796.

Horbaczewski: "Weitere synthetische Versuche über die Constitution der Harnsäure und Bemerkungen über die Entstehung derselben im Thierkörper." *Monatshefte für Chemie*, 1887, Bd. viii., S. 584.

Horbaczewski: "Beiträge zur Kenntniss der Bildung der Harnsäure und der Xanthinbasen, sowie der Entstehung der Leukocyten im Säugethierorganismus." *Monatshefte für Chemie*, 1891, Bd. xii., S. 221.

Huber: "Zur Verwerthung der Röntgen-Strahlen im Gebiet der inneren Medicin." Berliner Verein für innere Medicin. *Deutsche medicinische Wochenschrift*, 1896, p. 182.

Huber und Lichtenstein: "Ueber Gicht und ihre Behandlung mit Chinasäure." *Berliner klinische Wochenschrift*, 1902, No. 28.

Hutchinson: "The effect of muscular work upon the alkalinity of the blood." *Lancet*, 1896, p. 1166.

Hutchinson: "On lumpy induration of the skin of hands and feet in the descendants of gouty ancestors." *Edinburgh Med. Journal*, 1897, vol. 1. p. 289.

Jaccoud: "Du traitement de la goutte abarticulaire." *Semaine médicale*, 1895, vol. xv., p. 481.

Jaccoud: "Du traitement de la goutte articulaire aiguë." *Sem. méd.*, 1896, vol. xvi., p. 25.

v. Jaksch: *Ueber die klinische Bedeutung des Vorkommens von Harnsäure und Xanthinbasen im Blute der Exsudaten und Transsudaten*. Berlin, 1891, and *Zeitschrift für Heilkunde*, 1890.

Jolles: "Beiträge zur Kenntnis der Purinbasen." *Journal für praktische Chemie*, Neue Folge, 1900, Bd. xii., S. 61.

Jones: "Migraine and Arthritis." *Lancet*, 1898, vol. i., p. 326.

Kaufmann und Mohr: "Beiträge zur Alloxurkörperfrage und zur Pathologie der Gicht." *Deutsches Archiv für klinische Medicin*, 1902, Bd. lxxiv., S. 141, 348, 586.

Kionka: "Entstehung und Wesen der 'Vogelgicht' und ihre Beziehungen zur Arthritis urica des Menschen." *Archiv für experimentelle Pathologie und Pharmakolgie*, 1900, Bd. xliv., S. 186.

Kittel: "Ueber Uratablagerungen in der Fussohle, ihre Entstehung und Behandlung." *Berliner klinische Wochenschrift*, 1897, S. 358.

Kittel: *Die gichtischen Ablagerungen im menschlichen Körper*. 3. Auflage, Leipzig, 1902.

Klemperer, G.: *Untersuchungen über Gicht und harnsaure Nierensteine*. Berlin, 1896.

Klemperer, G.: "Zur Pathologie und Therapie der Gicht." Vortrag im Verein innere Medicin zu Berlin. *Deutsche medicinische Wochenschrift*, 1895, No. 40.

Klemperer, G.: "Ist Fischkost rathsamer als Fleisch bei harnsaurer Diathese." *Therapie der Gegenwart*, Neue Folge, 1901, Bd. iii., S. 428.

Kolisch: *Ueber Wesen und Behandlung der uratischen Diathese*. Stuttgart, 1895

Kolisch und Dostal: "Das Verhalten der Alloxurkörper in pathologischen Harnen." *Wiener klinische Wochenschrift*, 1895, Bd. viii., S. 413.

v. Kóssa, J.: "Künstliche Erzeugung der Gicht durch Gifte." *Archives internationales de Pharmacodynamie*, 1898, t. v., p. 97.

Kossel, A.: "Ueber Nucleïnsäure." *Centralblatt für die medicinischen Wissenschaften*, 1893, Bd. xxxi., S. 496; *Dubois' Archiv*, 1893, S. 157; 1894, S. 194.

Kossel und Neumann: "Ueber die Spaltungsproducte der Nucleïnsäure." *Sitzungsberichte der königlich preussischen Akademie der Wissenschaften*, 1894, Bd. xviii.

Krüger und Salomon: "Die Alloxurbasen des Harns." *Zeitschrift für physiologische Chemie*, 1898, Bd. xxiv., S. 364; Bd. xxvi., S. 350.

Krüger und Schittenheim: "Die Purinkörper der menschlichen Fäces." *Zeitschrift für physiologische Chemie*, 1902, Bd. xxxv., S. 153.

Krüger und Schmid: "Die Entstehung der Harnsäure aus freien Purinbasen." *Zeitschrift für physiologische Chemie*, 1902, Bd. xxxiv., S. 549.

Kühnau und Weiss: "Weitere Mittheilungen zur Kenntniss der Harnsäureausscheidung bei Leukocyten und Hypoleukocyten, sowie zur Pathologie der Leukämie." *Zeitschrift für klinische Medicin*, 1897, Bd. xxxii., S. 482.

Kuznitzky: "Aetiologie und Pathogenese der Psoriasis." *Archiv für Dermatologie*, 1897, Bd. xxxviii., S. 431.

Lange, O.: *Ueber Glaukom in seinen Beziehungen zu den Allgemeinerkrankungen.* Sammlung zwangloser Abhandlungen aus dem Gebiete der Augenheilkunde. Herausgegeben v. Vossius. Halle, 1896, Bd. i., Heft 6.

Laquer, B.: "Ueber die Ausscheidungsverhältnisse der Alloxurkörper im Harne von Gesunden und Kranken." *Verhandlungen des XIV. Congresses für innere Medicin*, 1896, S. 333.

Laquer, B.: "Ueber Beeinflussung der Alloxurkörperausscheidung durch Milchdiät und über Fettmilch bei Gicht." *Berliner klinische Wochenschrift*, 1896, No. 36.

Levison, F.: "Zur Behandlung der Gicht." *St. Petersburger medicinische Wochenschrift*, 1897 (Neue Folge xiv.), No. 1 und 2.

v. Leyden: *Verein für innere Medicin in Berlin*, 5 März, 1900.

Liebreich: Artikel "Colchicum" in *Encyklopädie der Therapie*, 1896, Bd. i., S. 757.

Lindemann: "Ueber locale Behandlung von Gelenksrheumatismus, Gicht, Ischias, etc., mit elektrischem Heissluftapparat (Elektrotherm)." *Therapeutische Monatshefte*, 1900, S. 117.

Lindner: "Onychogryphosis bei Gicht." *Deutsche Medicinalzeitung*, 1891, No. 65.

Loewi, Otto: "Beiträge zur Kenntniss des Nucleïnstoffwechsels." *Archiv für experimentelle Pathologie und Pharmacie*, 1901, Bd. xliv., S. 1.

Loewi, Otto: "Ueber die Stellung der Purinkörper im menschlichen Stoffwechsel." *Pflügers Archiv für die gesammte Physiologie*, 1901, Bd. lxxxviii., S. 296.

Ludwig: "Ueber den Einfluss des Karlsbader Wassers auf den Stoffwechsel," *Centralblatt für innere Medicin*, 1896, No. 45 und 46.

Luff, Arthur P.: "The gelatinous form of sodium biurate and its bearing on the pathology and treatment of gout." *Brit. Med. Journ.*, 28 Oct. 1899.

Luff, Arthur P.: "Modern views as to the causation and treatment of gout." *Lancet*, 1899, i., p. 1361.

Lülhje "Ueber Bleigicht und den Einfluss der Bleiintoxication auf die Harnsäureausscheidung. Inaugural-Dissertation. Berlin, 1895, *Zeitschrift für klinische Medicin*, 1896, Bd. xxix. (See also: Klemperer, *Untersuchungen*, 1896.)

Mgnus-Levy: "Harnsäure und Alkalescenz des Blutes in der Gicht." *Verhandlungen des XVI. Congresses für innere Medicin.* Wiesbaden, 1898.

Malfatti: "Ueber die Alloxurkörper und ihr Verhältnis zur Gicht." *Wiener klinische Wochenschrift*, 1896, Bd. ix, S. 723.

Malherbe et Legneu: "Des hématuries essentielles." *Gaz. hebdom.*, 1899, t. xlvi., p. 87.

Mayer, Paul: "Ueber den Einfluss von Nucleïn und Thyreoidinfütterung auf die Harnsäureausscheidung." *Deutsche medicinische Wochenschrift*, 1896, Bd. xxii., S. 186.

Mendelsohn, Martin: "Die Verschiedenheit des Problems der Harnsäureauflösung bei gichtischen Ablagerungen und bei Concretionen in den Harnwegen." *Deutsche medicinische Wochenschrift*, 1895, No. 18.

Mesnard: "Neurasthénie goutteuse." *Annales de la Policl. de Bordeaux*, 1897, v., p. 569.

Minkowski, O.: "Untersuchungen zur Physiologie und Pathologie der Harnsäure bei Säugethieren." *Archiv für experimentelle Pathologie und Pharmakologie*, 1898, Bd. xli., S. 375.

Minkowski, O.: Discussion zum Vortrage von His: "Das Verhalten der Harnsäure und ihrer Salze in Lösungen." *Verhandlungen des XVIII. Congresses für innere Medicin.* Wiesbaden, 1900, S. 438.

Minkowski, O.: "Die Ernährungstherapie bei harnsaurer Diathese, etc.," v. Leyden's *Handbuch der Ernährungstherapie*, 1899, Bd. ii., S. 499.

Minkowski, O.: "Ueber die Umwandlung der Purinkörper im Organismus." *Deutsche medicinische Wochenschrift*, 1902, No. 28.

Minkowski, O.: "Die Gicht." *Specielle Pathologie und Therapie* (Nothnagel), vii. Band, iii. Theil, 1903.

Lo Monaco: "Gli effetti della acque alcaline sul consumo azodato e sulla formazione dell' acido urico." *Policlinico*, 1896, iii., p. 345. (See *Schmidt's Jahrbücher*, 1897, Bd. ccliii, S. 124.)

Mordhorst: "Wirkungsweise des kohlensauern und des salicylsauren Natrons bei Gicht, Rheumatismus u. s. w." *Centralblatt für innere Medicin*, 1898, xix., p. 409.

Munter, S.: "Die Hydrotherapie der Gicht." *Blätter für klinische Hydrotherapie*, 1901, Bd. xi., S. 5.

Mylius: "Ueber die Einwirkung des Sidonals bei Gicht." *Therapeutische Monatsschriften*, December, 1900.

Myrtle: "Personal experiences of gout terminating in acute myositis of entire left lower extremity." *Brit. Med. Jour.*, June 9, 1900.

Neusser: "Ueber einen besonderen Blutbefund bei uratischer Diathese." *Wiener klinische Wochenschrift*, 1894, No. 39, S. 727.

Nicolaier: "Experimentelles und Klinisches über Urotropin." *Zeitschrift für klinische Medicin*, 1899, xxxviii.

Nicolaier und Hagenberg: "Ueber Chinotropin." *Centralblatt für Stoffwechsel- und Verdauungskrankheiten*, 1900, No. 6.

Nobécourt: "Rapports de l'intoxication saturnine et de la goutte." *Semaine médicale*, 1897, No. 7, p. 49.

v. Noorden: *Lehrbuch der Pathologie des Stoffwechsels.* Berlin, 1893.

v. Noorden: "Zur Behandlung der harnsauren Nierenconcremente." *Verhandlungen des XIV. Congresses für innere Medicin.* Wiesbaden, 1896.

v. Noorden: "Ueber Urol." *Centralblatt für Stoffwechsel und Verdauungskrankheiten*, September, 1901.

Offer und Roseqvist: "Ueber die Unterscheidung des weissen und dunklen Fleisches für die Krankenernährung." *Berliner klinische Wochenschrift*, 1899, No. 43 und 44.

Oliver: *Lead Poisoning.* Edinburgh and London, 1891.

Oppenheimer: "Gouty and rheumatic affections of the ear." *Med. News*, December 1898, lxxvii., p. 857.

Ortowski: "Vergleichende Untersuchungen über Urotropin, Piperazin, Lysidin, Uricedin und Natr. bicarb. bei der harnsauern Diathese." *Zeitschrift für klinische Medicin*, 1900, Bd. xl.

Oudin, Barthélemy et Béclère: "Diagnostic différentiel de la goutte et du rhumatisme chronique par la lumière de Roentgen." *Soc. médic. des Hôp.*, 28 Mai, 1897; *Semaine médicale*, 1897, p. 208.

Pel: "Die Erblichkeit der chronischen Nephritis." *Zeitschrift für klinische Medicin*, 1899, Bd. xxxviii., S. 127.

Pfeiffer: *Die Gicht und ihre erfolgreiche Behandlung.* Wiesbaden, 1891.

Pfeiffer: "Gichtfinger." *Berliner klinische Wochenschrift*, 1891, S. 369.

Pfeiffer: "Ueber die Ausscheiduugen im Urin während des acuten

Gichtanfalles mit besonderer Berücksichtigung der Harnsäure." *Berliner klinische Wochenschrift*, 1896, No. 15.

Poehl: "Einwirkung des Spermins auf den Stoffumsatz bei Autointoxicationen im Allgemeinen und bei harnsaurer Diathese im Speciellen." *Zeitschrift für klinische Medicin*, 1894, Bd. xxvi., S. 170.

Potain: "Des manifestations pulmonaires de la goutte." *Semaine médicale*, 1890, p. 41.

Potain et Serbanesco: "Radiographie des extrémités chez les sujets affectés de goutte ou de rhumatisme chronique." *Académie des Sciences*, 18 Juin, 1897.

Pribram: "Chronischer Gelenksrheumatismus und osteoarthritis deformans." *Nothnagel's Handbuch*, 1902, Bd. vii.

Proust: "Du régime alimentaire du goutteux." *Progrès médical*, 1896, No. 32.

Richter, Paul Fr.: "Ueber die experimentelle Prüfung sogenannter 'Gichtmittel' im Allgemeinen und über die Chinasäure und das chinasaure Piperazin im Besonderen." *Charité-Annalen*, 1900, Bd. xxv., S. 197.

Roberts, W.: *On the chemistry and therapeutics of uric acid, gravel, and gout.* London, 1892.

Salfeld: "Zur Behandlung der Gicht mit Chinasäure." *Münchner medicinische Wochenschrift*, 1901, No. 16.

Salomon und Krüger: "Die Alloxurbasen des Harnes und ihre physiologische Bedeutung." *Deutsche medicinische Wochenschrift*, 1899, No. 6.

Schott: "Ueber gichtische Herzaffectionen und deren Behandlung." *Verhandlungen des XIV. Congresses für innere Medicin*, 1896, S. 486.

Senator: "Ueber die Unterscheidung des weissen und dunklen Fleisches für die Krankenernährung." *Berliner klinische Wochenschrift*, 1899, No. 45 und 49.

Singer: "Ueber Aspirin." *Pflüger's Archiv*, 1901, Bd. lxxxiv., S. 527.

Smith-Jerome: "The formation of uric acid in man and the influence of diet in its daily output." *Journal of Physiology*, 1897, vol. xxii., p. 146.

Smith-Jerome: "Further proofs of the origin of uric acid from nuclein compounds and derivatives." *Journal of Physiology*, 1899, vol. xxv.

Sternfeld: "Die Chinasäure, ein neues Heilmittel gegen Gicht." *Münchener medicinische Wochenschrift*, 1901, No. 7.

Strauss, H.: "Pathogenese und Therapie der Gicht im Lichte der neueren Forschungen." *Würzburger Abhandlungen*, 1902, ii., 8.

Strümpell: "Ueber Vorkommen und Diagnose der Gicht." *Münchener medicinische Wochenschrift*, 1900, No. 38.

Surmont et Brunelle: "Recherches sur l'élimination de l'azote urinaire au cours et dans la convalescence de la colique saturnine." *Archiv. génér. de Méd.*, 1894.

Teissier: "Goutte," In Brouardel, Gibert et Girode, *Traité de médecine*, 1897, t. iii

Watson, Chalmers: Article "Gout," in *Encyclopædia Medica*, 1901, vol. iv.

Weintraud, W.: "Beiträge zum Stoffwechsel in der Gicht." *Charité-Annalen*, 1895, xx., S. 215.

Weintraud, W.: "Ueber Harnsäure im Blute und ihre Bedeutung für die Entstehung der Gicht." *Wiener klinische Rundschau*, 1896, x., No. 1 und 3.

Weintraud, W.: "Die Gicht." *Ergebnisse der allgemeinen Pathologie* von Lubarsch und Ostertag. Wiesbaden, 1896, Bd. i.

Weiss, J.: "Die Chinasäure als Antiarthriticum." *Verhandlungen der Deutschen Gesellschaft der Naturforscher und Aerzte*, München, 1899.

Wilcox: "A phase of the treatment of goutiness." *Medic. News*, Nov. 27, 1897.

Zagari e d'Amato: *Influenza delle inalazioni di ossigeno e del moto sull' eliminazione dell' acido urico e corpi affini.* Napoli, 1898.

Zagari e Pace: *La genesi dell' acido urico e la otta*, 1897.

AMENTIA.

IDIOCY—IMBECILITY—FEEBLE-MINDEDNESS.

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(With Plates II. to XI.)

THE term amentia comprehends all the varieties and degrees of mental abnormality due to imperfect or arrested development, which are popularly known as idiocy, imbecility, and feeble-mindedness. The condition must be clearly distinguished on the one hand from insanity—the abnormal action of a fully-developed mind, and on the other from dementia—failure of the mental powers due to degenerative processes; such a distinction is particularly necessary since the milder grades of the latter infirmity are frequently referred to as imbecility or feeble-mindedness. The practitioner is often consulted with regard to mentally-defective children, and on that account it is hoped that a general survey of the degrees, chief varieties, diagnosis, prognosis, and main lines of treatment of this condition may be not without value.

The incompletely-developed mind, which is the essential characteristic of amentia, may be due to one of two causes, viz.:—(1) to an inherent incapacity for perfect development owing to defects within the embryonic rudiment; this is responsible for about 90 per cent. of all cases, and the condition is designated *primary amentia;* or (2) to the mental development having been arrested by some extraneous or accidental cause; this accounts for the remaining 10 per cent. of cases, and the condition is called *secondary amentia.*

Without going into ætiological details,[1] it may be remarked that the factors bringing about the imperfect development of the primary form are morbid ancestral conditions, especially insanity or epilepsy, tuberculosis and alcoholism; whilst those producing the arrest of development met with in the secondary form are usually traumatic (including asphyxia neonatorum) or toxic processes. The two groups thus differ entirely from one another with regard to their causation and pathology, and also, as we shall subsequently see, in their clinical features and prognosis. It is, therefore, important clearly to distinguish between them. It is desirable to state that the terms "congenital" and "acquired," as used with reference to the birth of the patient, are not quite synonymous with those here given, since a true secondary amentia may result from some ante-natal, though extrinsic, condition, and occasionally inherent defects require some slight morbid process or physiological stress after birth to bring them into prominence. I think, therefore, that the words *primary* and *secondary* are not only more accurate, but enable us better to realise the true features and differences of these two chief divisions.

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[1] The ætiology of amentia is fully discussed by the writer in Mott's *Archives of Neurology,* Vol. II., 1903.

PRIMARY AMENTIA.

In this group the essential condition is one of imperfect development, which is due to defects inherent in the germinal plasm, the result of morbid ancestral influences. In the majority of the cases the mental condition is only part, although perhaps the most important part, of an incompleteness and inferiority widespread throughout the organism, as is fully shown by the presence of numerous physical defects and abnormalities, called "stigmata of degeneracy." In the milder cases the mental faculties only, and perhaps even but the highest of these, are affected.

The pathological condition is generally as follows: the brain may vary greatly in size and external appearance, but on the whole is smaller and more simply convoluted than normally; the cortical cells are fewer, irregularly arranged, and many of them imperfectly developed, and the horizontally-coursing bundles of association-fibres are strikingly diminished when compared with the healthy brain. There may in addition be gross pathological lesions or developmental anomalies, but on the whole the extent of the changes above described is directly proportionate to the amount of mental deficiency observed during life.

Such are the characteristics common to all cases of primary amentia; but if we examine a sufficiently large collection of these patients we notice that a small percentage of them possess such special peculiarities of cranial conformation and general physiognomy, frequently associated with particular mental qualities, that they are readily set aside as distinct types. The group of primary amentia may, therefore, be conveniently divided into the following chief varieties:—

1. *Simple.*
2. *Microcephalic.*
3. *Mongolian.*

In addition to the above, *Scaphocephalic*, *Negroid*, *Grecian*, and *American-Indian* varieties have been described, but they are not sufficiently common to be specially referred to here.

1. *Simple.*

The great majority of cases of primary amentia possess no distinguishing physical characteristics beyond the stigmata of degeneracy common to the whole group; they may, therefore, be termed the simple or ordinary variety. The term "genetous" is used by Ireland for these patients, but is open to the objection[1] that *all* primary aments are in reality genetous.

It is obvious that in any variety the *degree* of mental defect may vary within very wide limits, and probably in no type are these differences greater than in the one we are now considering, where the mental defect may range from the grossest idiocy to a subnormality so slight as to be almost imperceptible. The question of degree, therefore, is of great importance, and it is necessary to guard against the common error of confounding this with the variety. The Simple variety, then, may be divided into three degrees of (A) *Feeble-mindedness*, (B) *Imbecility*, (C) *Idiocy*, a purely arbitrary division, since there is no hard-and-fast line between them, but convenient for purposes of classification.

It may be remarked that the attempt has been made to make the word "feeble-mindedness" a generic term synonymous with amentia, and indeed in America it has this application; but in this country it is used specifically to indicate the mildest degree of mental defect.

A. *Feeble-mindedness.* (Plates II. and III., Figs. 1, 2, 3, 4.)

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[1] The word is also extremely ugly and ill-formed.—ED.

PLATE II.

Primary Amentia—Simple Variety—High Degree (Feeble-mindedness).

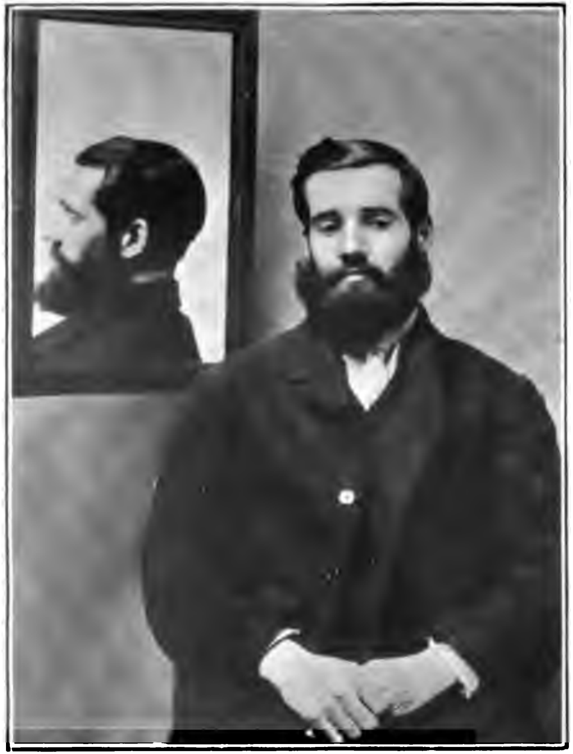

Fig 1. *A patient of the emotional religious type, suffering from a slight degree of feeble-mindedness. He is very unstable, and has had several attacks of insanity ("religious mania").*

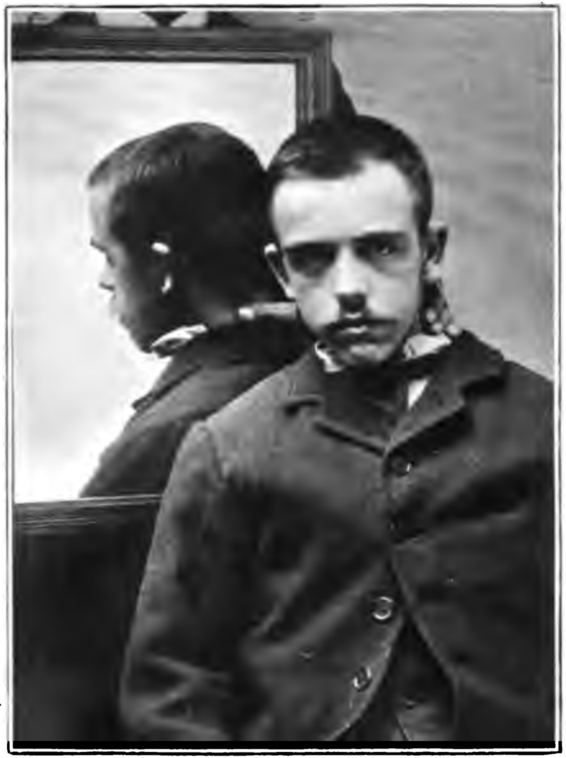

Fig. 2. *A feeble-minded youth of the surly, loafing type. No regular employment, firmly convinced that he has "not had fair play." Has had attacks of insanity (acute melancholia).*

PLATE III.

Primary Amentia—Simple Variety High Degree (Feeble-mindedness).

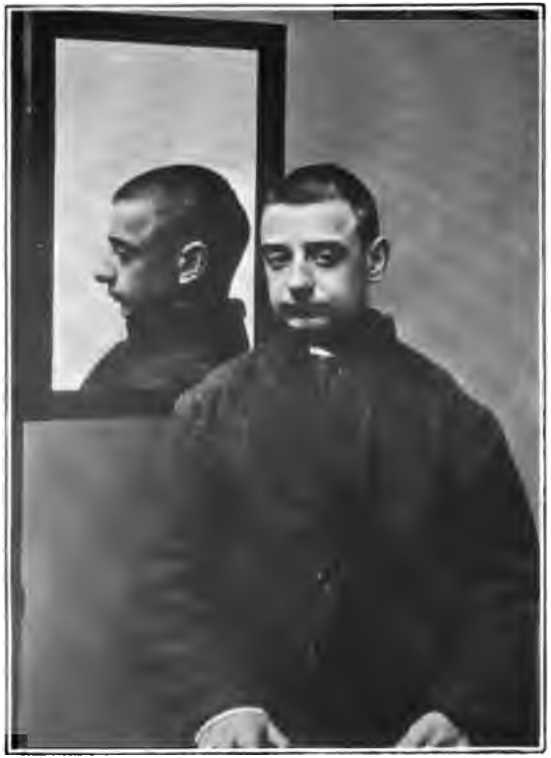

Fig. 3. *A feeble-minded youth of the criminal type. Lacking in judgment and power of attention. Restless, idle, untruthful, and roving disposition. Was imprisoned for theft before he was sent to the asylum.*

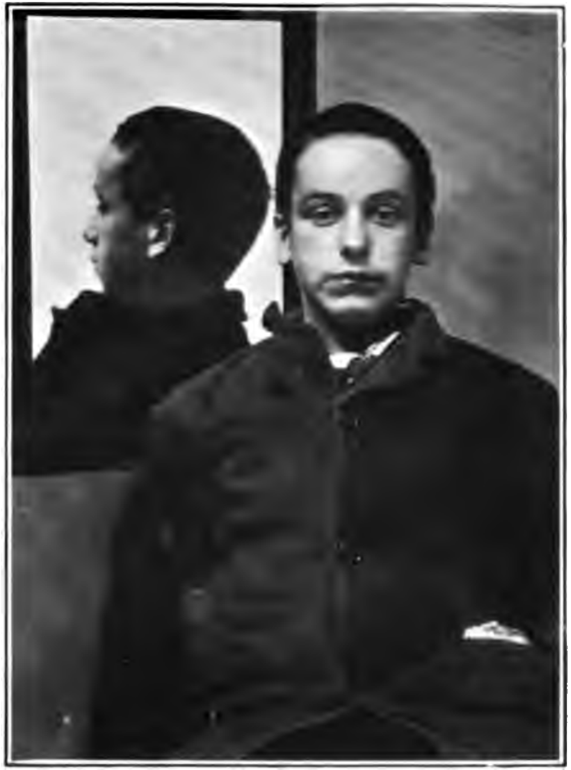

Fig. 4. *A youth suffering from more pronounced degree of feeble-mindedness. Will not work and has very little power of initiation.*

—This class is composed of patients suffering from the mildest degree of mental deficiency; nevertheless its members are far from being of one uniform standard of intelligence, since in this, as in other degrees, there is a regular series of gradations, The higher members of the feeble-minded approach very closely to the individual of normal mental ability, indeed in some of these patients it may need all the experience of an expert to determine whether mental deficiency is present or no. The condition of many members of the loafing and criminal classes is in all probability really the result of a subnormal intellect, whilst cases of so-called "moral imbecility" form another connecting link between the normal population and the feeble-minded as ordinarily understood. Many cases of "adolescent" insanity also belong to this class. Other patients suffering from a mild degree of mental feebleness may possess really extraordinary talents in certain directions, *e.g.*, mechanical or musical, although at the same time they may not have sufficient common-sense to take care of their own interests without help. These latter, however, form the show cases of institutions, and are rather the exception than the rule; and in the majority of the feeble-minded there is a well-marked and obvious mental defect, an inability to progress, in mechanical work as well as in learning, at the same rate as their compeers, so that special education and some amount of permanent supervision are absolutely necessary. On enquiry it will be found that these patients have nearly always been backward from a very early age, that in learning to walk and talk they have been behind average children, although usually their condition does not attract the serious concern of their parents until school life is begun. Then, instead of passing on from class to class, they remain fixed in the lowest, and one of the commonest statements heard with regard to these patients is that they could never get on at school. Dr. Warner has estimated that such children comprise one per cent. of the school population.

The Defective and Epileptic Children's Act (62 & 63 Vict., ch. 32, 1899) has recognised the difficulties of educating these children who, "not being imbecile, and not being merely dull or backward, are incapable of receiving instruction in the ordinary elementary public schools," and has provided for the establishment of special classes and schools to meet their requirements.

Feeble-minded children may differ but little from those of average intelligence in their general appearance, many of them being well-nourished and well-grown, active and fond of play and games, and often having a very good memory for the things they do learn. In the larger number of them, however, indications of degeneracy and of abnormal nervous action will be found, and the lower members of the group in their mental and physical conditions approximate to the imbeciles. Usually an inability to concentrate their attention upon any subject which is uncongenial to them is present in these patients; they also have little imagination and power of ideation, their association of ideas is imperfect, and their faculties of judgment, comparison, and control are very poorly developed. As a result of suitable training a not inconsiderable number become sufficiently adept at various kinds of handywork to support themselves partially, or even entirely, but it is very rarely that they can hold their own in the world without supervision. They lack pertinacity, and in consequence seldom retain a situation for long, if they are free to go where they please. One girl whom I knew, who was in domestic service, changed her situation no less than fourteen times in less than three years. In addition many of them are mentally unstable, although mental defect is not necessarily synonymous with mental instability, and many, perhaps even the majority, of the feeble-minded are placid, good-tempered, and industrious to the end of their days. Where such instability exists, however, great care and watchfulness should be exercised, not only in the interests of the patient, but also of those around them, lest in a sudden outburst of violence or fit of moroseness injury should be inflicted on little children, or suicide attempted. Actual insanity indeed frequently occurs in the unstable feeble-minded and also in the higher-grade imbeciles, in consequence of which removal to an asylum may be imperative.[1] Epilepsy also is not an uncommon accompaniment of these cases, but paralysis and indications of coarse brain-lesion are rare, since, when these exist, the mental defect is generally more pronounced.

[1] *Vide* article "Insanity in Imbeciles," *Journal of Mental Science*, January, 1903.

PLATE IV.

Primary Amentia—Simple Variety—Medium Degree (Imbecility).

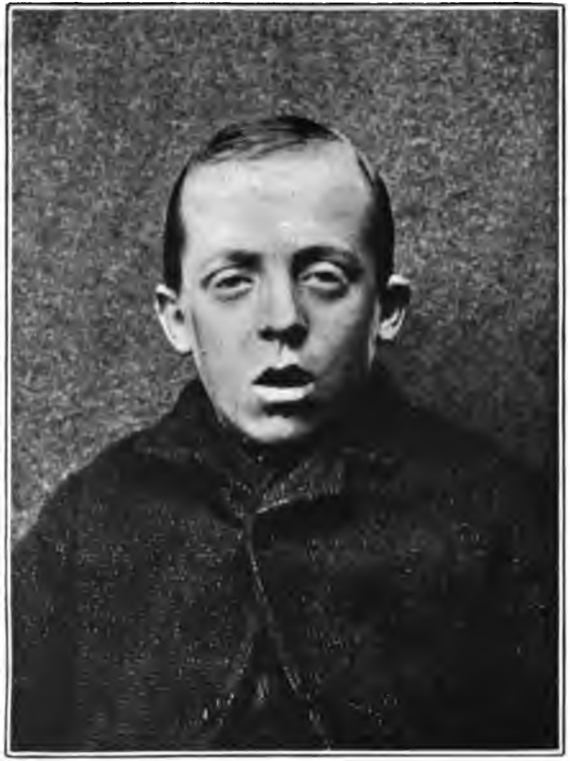

Fig. 5. *Imbecile of mild grade. A few stigmata of degeneracy. Can do some work under supervision.*

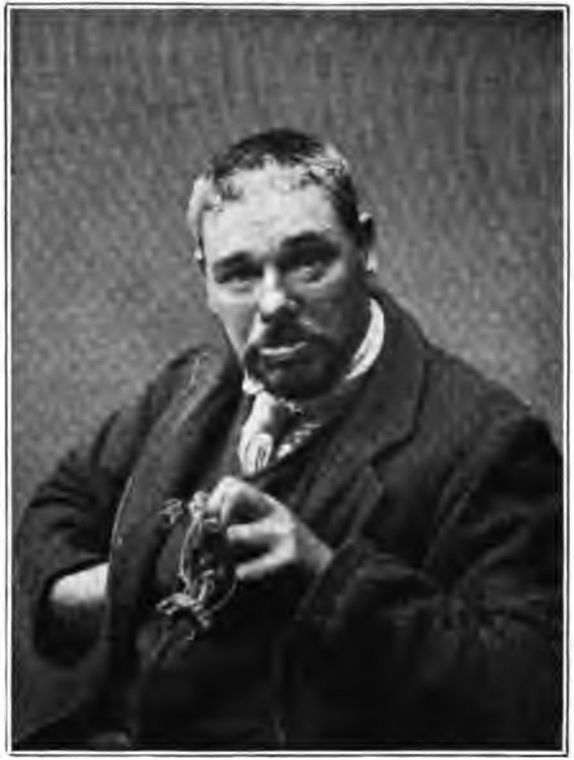

Fig. 6. *Imbecile of low grade. Idle, restless, mischievous, and full of tricks.*

PLATE V.

Primary Amentia—Simple Variety—Low Degree (Idiocy).

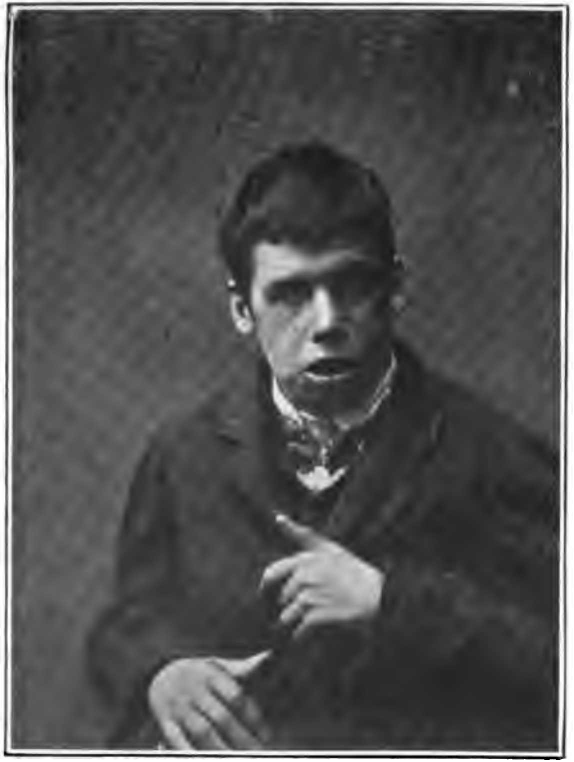

Fig. 7. *Idiot. Unable to speak, dress, feed, or keep himself clean. Possesses instincts of self-preservation. Spends the greater part of his time sucking his thumb.*

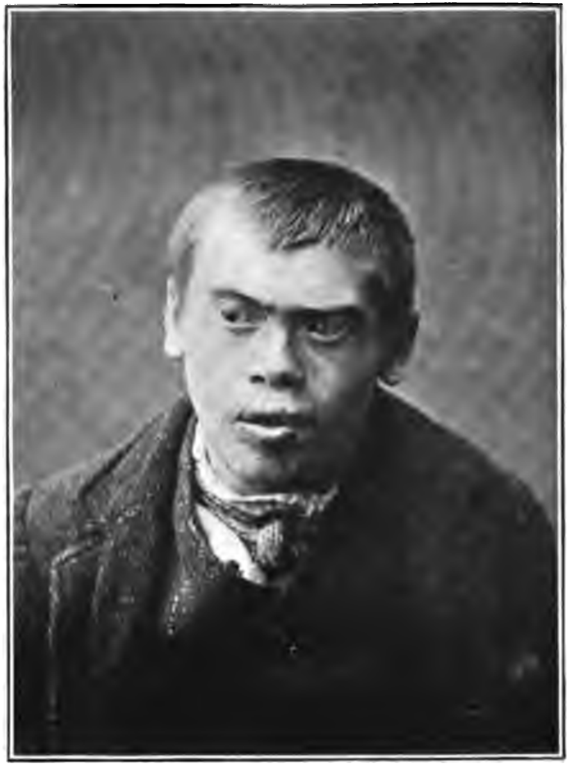

Fig. 8. *Idiot, with marks of congenital syphilis. Unable to speak, dress, feed, or keep himself clean.*

B. *Imbecility*. (Plate IV., Figs. 5 and 6.)—Although the law distinguishes between the mild degree of amentia just considered and that of imbecility, in excluding the latter from the provisions of the Defective Children's Act above referred to, and requiring their certification under the Idiots' Act, yet scientifically there is no hard-and-fast line between them, and the one degree gradually merges into the other; indeed it is often an extremely difficult matter to decide whether the infirmity of a particular patient is or is not such as to debar him from the special classes for the feeble-minded. The imbecile labours under the same deficiency of control, of judgment, and of comparison, the same imperfect association of ideas and inability to concentrate the attention, although to a greater extent. As we pass down the scale, however, we notice an additional defect in the lower mental faculties; there is a lessened power of storing impressions, less retentiveness, although there may be no defect in the sensory channels; but in consequence of this the response to education is much less satisfactory. It therefore follows that the most practical means by which these two classes can be distinguished from one another is found in their capability of responding to education, and the practitioner will be wise to refrain from certifying a case of supposed imbecility which is not sufficiently obvious until this test has been applied. Certification may lead to the incarceration of the patient in a county asylum, where suitable training is impossible, whereas the educational methods of a special class for the feeble-minded might result in great improvement.

With regard to the lower degrees of imbecility there will seldom be much difficulty, since in their pronounced and obvious mental defect and in their stigmata of degeneracy they approximate to the idiots. In these lower grades epileptic fits and various forms of paralysis are of common occurrence, and I have already remarked that insanity is by no means infrequent in the higher grades.

C. *Idiocy*. (Plate V., Figs. 7 and 8.)—Perhaps the most convenient criterion by which this class may be distinguished from that just described is furnished by the faculty of speech, since, although a few idiots may be able to repeat short words, or even sentences, in parrot-fashion, true speech is usually wanting. As an instance, however, of the impossibility of relying upon any one sign to differentiate the degrees of amentia, it must be remembered that occasionally quite high-grade imbeciles are deficient in the power of articulation.

In idiocy, in addition to the defects already noticed in the two milder grades, we find other and more gross imperfections of both body and mind. Physical deformities and stigmata of degeneracy are very numerous, and the whole vitality of the child is diminished to such an extent as to render him prone to many forms of disease, and often to early death. On the mental side also, not only is there deficiency in the higher faculties, and a diminished power of retaining impressions as in the imbeciles, but generally, too, a lessened ability actually to *perceive,* which applies to almost all the sensory channels. Sight and hearing are very imperfect; the idiot often appears to have practically no sense of taste or smell, and will, consequently, eat the most disgusting garbage, whilst in many cases he is practically indifferent to extremes of heat or cold. In my opinion it is this deficiency of perception which is the chief psychological characteristic of the idiot as distinguished from the imbecile.

There are several degrees even of idiocy, ranging from that in which the patient can be taught to feed, wash and dress himself, and to obey simple commands, to that of the hideously-repulsive creature unable to walk or stand, with no instincts of self-preservation, simply respiring, digesting, and excreting, who would die of cold and hunger if not kept warm and plied with food. Many of these patients are simply inert masses of degenerate humanity, others are in an incessant state of automatic activity, whilst yet others fill the air of the ward with ceaseless yells and screeches, making it a veritable pandemonium. The lower members of this grade constitute the absolutely incurable residue of the order of Amentia.

2. *Microcephalics* (Plate VI., Figs. 9 and 10).

About 6 per cent. of primary aments belong to this variety. It is usually considered that the criterion of the condition is a skull with a circumference of less than 17 inches; but as a matter of fact mere size cannot alone be depended upon to

PLATE VI.

Primary Amentia—Microcephalic Variety.

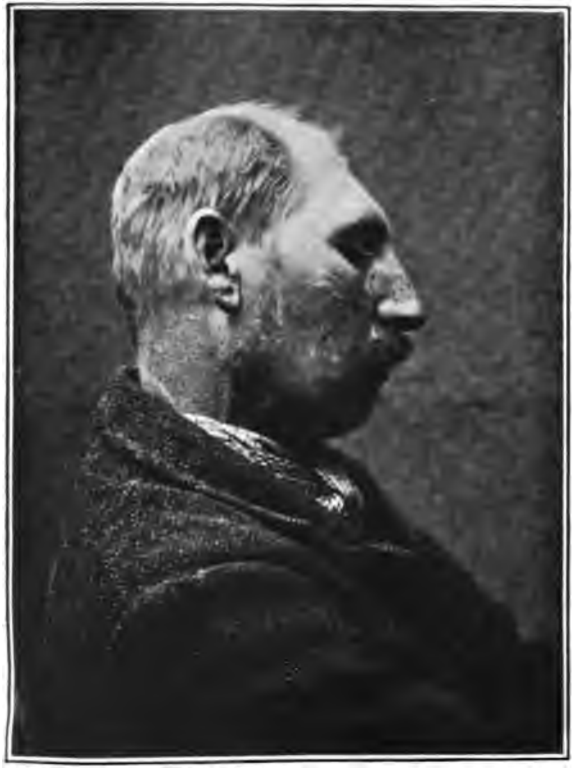

Fig. 9. *Medium degree of amentia (imbecile).*
(Cranial circumference, $18\frac{7}{8}$ *inches.)*

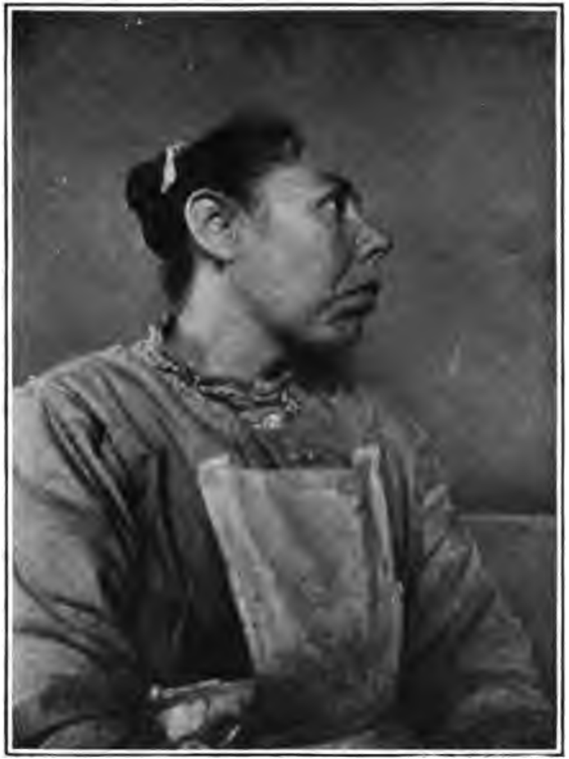

Fig. 10. *Medium degree of amentia (imbecile).*
(Cranial circumference, 17 *inches.)*

distinguish the class, the distinctive characteristic being rather a peculiarity of *shape*. The accompanying figures will give a better idea of these patients than any description, and if the condition is once seen it can hardly be again forgotten. Microcephalics usually come of a pronounced neuropathic stock; their brothers and sisters, as a rule, are typical degenerates, and frequently one or more of them also suffer from microcephaly. The condition of the brain is one of general hypoplasia, and the hemispheres are often so imperfectly developed, especially in the temporo-sphenoidal and occipital regions, as to leave a portion of the cerebellum uncovered, when the brain is removed from the skull. Coincidently with this, the convolutions are comparatively few and simple in arrangement, and often occupy abnormal positions, whilst the secondary gyri may be wanting altogether. The entire brain may weigh no more than 200 grammes. Microscopical examination shows the nerve-cells to be very few in number, and imperfectly developed; but whereas in the Simple variety this paucity chiefly concerns the smaller and medium-sized pyramids, in the Microcephalics the large pyramids and Betz' cells are also often diminished. As a result of this, the motor tracts in the cord are very poorly developed; indeed, to the naked eye they may appear almost absent, and even the fibres of the posterior columns and the cells of the anterior horns may be less plentiful than usual.

This characteristic conformation of brain gives rise to a corresponding peculiarity of cranial configuration, and results in the grotesque appearance which is so typical of these patients. There is a marked deficiency in the frontal and post-parietal regions, so that the whole cranium shelves away to a summit almost like a sugar-loaf, appearing strangely out of proportion to the normally-sized face underneath.

Another rather characteristic feature of these patients is their extraordinarily thick scalp with coarse wiry hair; on several occasions I have known the teeth of the clipper to be actually broken whilst the hair was being cut. Occasionally the scalp is thrown into four or five deep furrows running in an antero-posterior direction, a condition to which attention was first drawn in this country by Dr. McDowall, of Morpeth Asylum.

There is now no reason for thinking, as was formerly believed, that the deficient growth of the brain is due to premature union of the cranial sutures, since many cases of microcephaly have been recorded in which these were *not* closed. When premature synostosis is present, it is to be looked upon as the result, rather than the cause, of the imperfect cerebral development; hence operations directed towards increasing the cranial capacity are useless.

It has also been sought to show that these patients are examples of atavism, and much has been written with the object of showing the simian or even lower characteristics of their brains. Undoubtedly in a lower stage of our existence the brain was a far less complicated organism than it is now, but that microcephaly is not simply a reproduction of this stage is shown by the fact that the lower neuronic systems (motor and sensory) are involved at the same time as those of a higher order. In the imperfections of development which result from disease we have not simply a putting back of the hand of time, but other processes are involved, which we do not yet understand; and the microcephalic, although an imperfect, is also a degenerate human being.

As a result of the deficiency of the motor tract there is often a general helplessness—it can hardly be called paralysis—which makes walking a late acquirement and performed with difficulty. This extreme helplessness is usually associated with a pronounced degree of amentia, but other patients may be much more active in mind and body. In the medium grades of microcephaly there is frequently considerable power of observation and memory, and these patients possess no small imitative faculty, so that if their energy can be directed into useful channels they may be taught to run errands and do various kinds of manual work not requiring much intelligence. Also I have often seen patients with a typical microcephalic head and the characteristic activity and imitativeness, who were only feeble-minded. As a rule, perhaps, the degree of intelligence can be roughly gauged by the cranial circumference; but this test is by no means infallible. Nearly half of these patients suffer from epileptic fits, and although a few of the milder cases may attain middle or even fairly advanced age, the majority of the pronounced cases succumb either to

Plate VII.

Primary Amentia—Mongolian Variety.

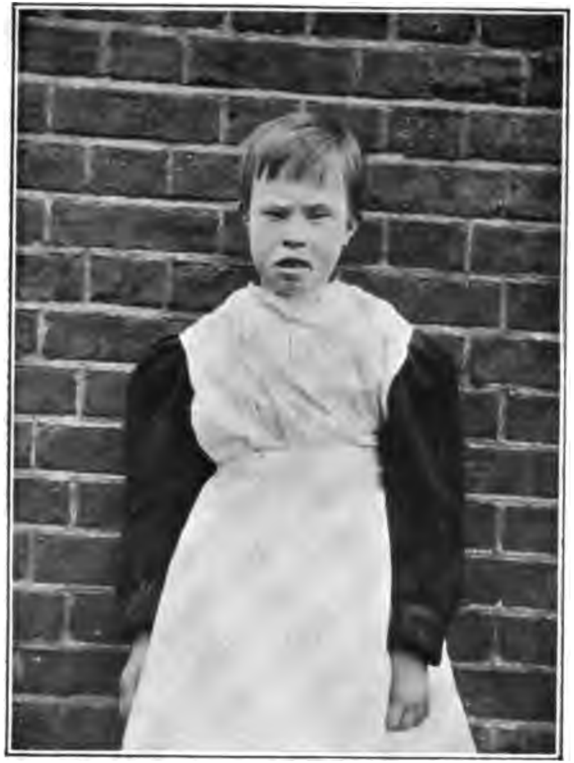

Fig. 11. *A Mongolian ament of medium grade.*

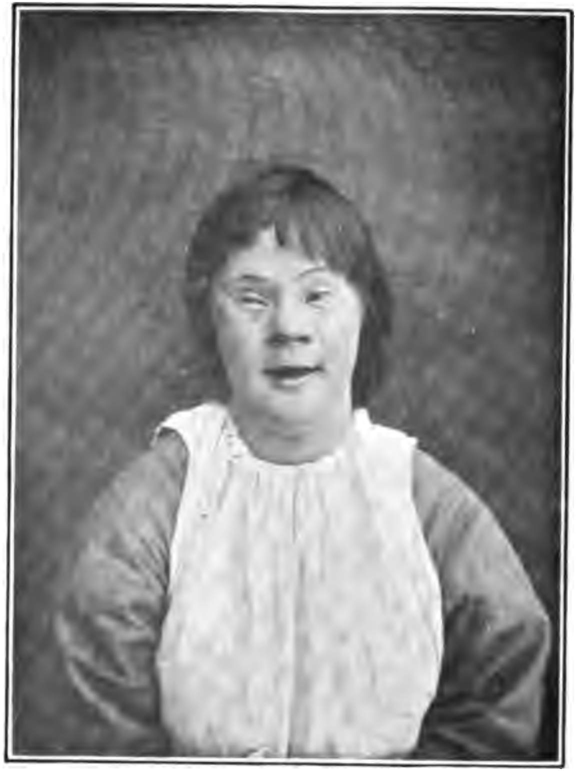

Fig. 12. *A Mongolian ament of low grade.*

consumption or some inflammatory condition of the brain in early life.

3. *Mongolians* (Plate VII., Figs. 11 and 12).

The members of this variety are not very numerous, and probably do not form more than about 3 or 4 per cent. of all cases of amentia; their characteristics, however, are very distinctive. It has been suggested by Dr. Sutherland[1] that the condition is a para-syphilitic one, but my own experience does not confirm this view. Through the courtesy of Dr. Taylor, I had the opportunity of examining a considerable number of Mongolians of varying degrees at Darenth Asylum, and also of making a searching enquiry into their family history, and I entirely failed to find evidence of any preponderance of syphilis. On the other hand there was nearly always a well-marked neuropathic history, and frequently also a strong tubercular taint; in addition, the children were usually the later-born of a large family, which often numbered as many as ten or twelve. I therefore think that the condition is simply and solely due to those devitalising and degenerating influences which are the cause of primary amentia in general, although for some reason or other the imperfection has run in particular lines, and thereby given rise to a distinct variety.

In these patients the cerebral hemispheres are small and simply convoluted, but in addition the medulla, pons, and especially the cerebellum, are also reduced in size; it is probable that it is on account of these imperfections in the latter structures that the base of the skull does not develop to its ordinary size, thus causing the whole cranium to be small and rounded, and producing those Mongolian peculiarities of physiognomy which are the distinguishing features of this variety, *i.e.*, diminutive palpebral fissures, directed downwards and inwards, with flattening and broadening of the nose. Besides this the tongue is frequently so large that it causes the mouth to be partly open, and it is usually marked by numerous transverse fissures and prominent papillæ; the hair is wiry, the skin coarse, the hands and feet short and stumpy, and the little finger often incurved in a characteristic manner.

As a rule in these patients the amentia is only of a medium grade, most of them being cheerful, pleasant, harmless, very imitative, and curiously fond of music. Many of them may be made fairly useful in many ways, although the clumsy condition of their hands prohibits any operation requiring manual dexterity. It is a singular fact that paralysis and fits are extremely uncommon amongst them; indeed I have only seen two of these patients who were subject to epilepsy. On the other hand they are very prone to catarrhal affections, and sore eyelids, cracked lips, and chronic rhinitis are exceedingly prevalent amongst them. Congenital heart-lesions also occur in a very large proportion, due to the persistence of fœtal structures, and it is owing to these or to some catarrhal or tubercular process that the majority die early, few Mongolians reaching the age of 30 years. There will seldom be any difficulty about the diagnosis of a case presenting the above definite signs; indeed, it is often possible to recognise the condition during the early months of life. Occasionally, however, one sees patients possessing but a few of these features who seem to be intermediate between the Mongolian and Simple varieties.

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[1] PRACTITIONER, December, 1899.

The Complications of Primary Amentia.

It has been pointed out that the types or varieties just described are simply due to differences of general physiognomy and of cranial conformation, and it is probable that such differences are the result of the greater or lesser incidence of the injurious ancestral influences upon some particular portion of the primary nervous rudiment, although as to why there should be any such selection we as yet know nothing. It is now necessary to observe that in each of these chief varieties of imperfect development we may have other conditions accompanying and complicating them. These complications are divisible into two classes: (A) *Gross Anomalies of Development;* (B) *Secondary Morbid Processes.*

The former group comprises such conditions as *microgyria, localised agenesis, true porencephaly, diffuse or circumscribed sclerosis,* and many other malformations; whilst in the latter we have *hæmorrhage, thrombosis, meningo-encephalitis, hydrocephalus, false porencephaly, cysts,* &c. With regard to the anomalies of development much has been written in the

PLATE VIII.

Primary Amentia—Simple Variety—Low Grade—Complicated by Sclerosis.

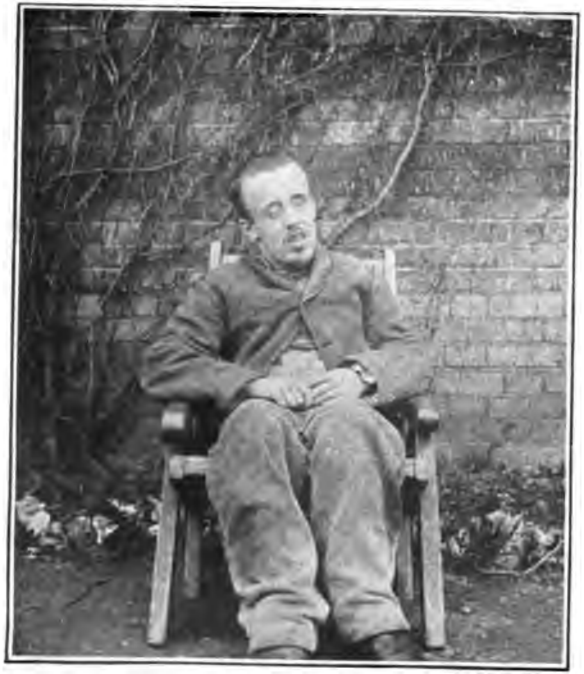

Fig. 13. *A case of so-called "hypertrophy of the brain," showing the characteristic cranial enlargement with numerous stigmata of degeneracy.*

Primary and Secondary Amentia.

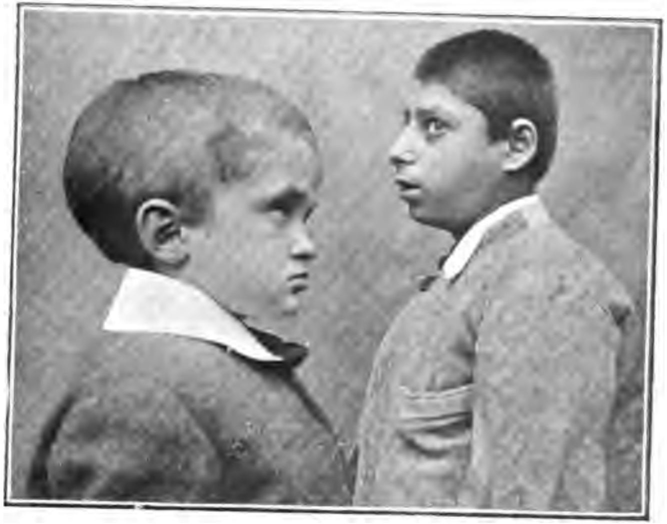

Fig. 14. *A case of primary amentia of the microcephalic variety, and one of secondary amentia due to a cerebral lesion (hydrocephalus).*

attempt to associate them with particular forms of mental defect, and indeed to consider them as the *fons et origo* of mental deficiency generally. It is, of course, true that agenesis, &c. of some portion of the cerebrum concerned with special functions will probably lead to defect of those functions, but to look upon these abnormalities as the actual cause of the amentia is to my mind utterly erroneous, and it would be just as logical to attribute the poor vitality and general ill-health and proneness to disease of these patients to their stigmata of degeneracy; both are simply a further and more gross manifestation of cellular imperfection.

With regard to the second group, we shall see in discussing secondary amentia that pathological processes unaided may sometimes produce arrest of mental development in a previously healthy brain, but in the majority of cases of amentia they also, in my opinion, are merely complications, and not the actual cause. The imperfectly-developed brain seems more than usually liable to such morbid processes, which perhaps is scarcely surprising.

These complications cannot always be diagnosed during life, and I have more than once found *post mortem* severe hydrocephalus in a microcephalic idiot, or agenesis, porencephaly, sclerosis, and other developmental anomalies, where none had been suspected. Since, however, brain-lesions often give rise to definite symptoms their existence may at times be conjectured or even definitely affirmed. Thus some degree of unilateral paralysis, especially if accompanied by imperfect development of the paralysed limb, is suggestive of porencephalus; if epileptic convulsions are also present a meningo-encephalitis is probable. Hydrocephalus is usually recognised by the characteristic cranial enlargement, but even where this is absent, its presence may be suggested by a gradually increasing spasticity and progressive dementia; whilst sclerosis (sometimes called hypertrophy of the brain) is often distinguished by a large head of characteristic shape, somewhat resembling hydrocephalus, and a tendency to convulsions in series, usually without paralysis (Plate VIII., Fig. 13). It is important to bear in mind that the presence of one of these lesions offers a material impediment to amelioration of the amentia, and, therefore, considerably affects the prognosis.

It is impossible to state what proportion of patients suffering from primary amentia are affected by either or both of these kinds of complications—the milder degrees probably rarely, the idiots I believe with extreme frequency—but since any of the varieties may be so affected it seems more desirable that they should be considered simply as complications, or, at the most, as subvarieties, rather than as denoting specific types.

SECONDARY AMENTIA.

The cases we have now to consider, although probably not comprising at the most more than 10 per cent. of all aments, are of an entirely different nature ætiologically, pathologically, and clinically from those hitherto dealt with. Here there is no morbid influence at work upon the germinal plasm of sufficient intensity to interfere with mental development—on the contrary, in many cases the family history is particularly good—and the amentia is merely a symptom of some entirely extraneous, and in most cases accidental, condition. There is, nevertheless, every gradation of mental deficiency to be found amongst them, just as in the primary group, and we consequently see patients of this class who are idiots and others who are imbecile or simply feeble-minded, although probably the greater number of them are of the medium grade. The description which has already been given of the psychological status of these three degrees will, therefore, equally apply to the cases we have now to consider, though, as will subsequently be pointed out, there is a very great difference in the appearance of the two groups.

Whilst cases of primary amentia are all the result of one factor—morbid heredity—those of secondary amentia result from many and diverse conditions, so that it is convenient to divide them into four different ætiological groups, which I propose briefly to describe. They are as follows :—

Amentia due to
- (A.) *Gross Cerebral Lesions.*
- (B.) *Nutritional Defect.*
- (C.) *Special-Sense Defect.*
- (D.) *Degenerative Processes.*

(A.) *Amentia due to Gross Cerebral Lesions.*

The cases in this group may be considered as the type *par excellence* of secondary amentia, the condition being solely and simply the result of a fortuitous, though unfortunate, circumstance. The patients are perfectly free from any neuropathic inheritance, and until their mental development is suddenly brought to a standstill are in every way normal; consequently there are no stigmata of degeneracy, and even the lower grades may be comely and physically well-developed. The cause of the arrest is in every case a localised lesion of the brain, which may be produced by many different agencies, but which are broadly divisible into two classes, viz., (A.) *Vascular* (i.e., *thrombosis* or *hæmorrhage*), the result of asphyxia neonatorum, trauma, whooping-cough, &c.; and (B.) *Toxic*, which may be part of a general infection like scarlet-fever, measles, small-pox, typhoid fever, &c.; or due to the extension of a hitherto localised otitis, rhinitis, or meningitis; or occasionally a direct primary infection of the nerve-cells.

Now it is probable that the number of children in whom cerebral complications occur as a result of one or other of the above causes is not inconsiderable, and a large proportion of those affected die; others, but only a few, appear to recover completely; in a few more death does not take place, but a permanent legacy remains in the shape of a gross cerebral lesion, and in the majority of these some degree of mental deficiency results.

In the cases of the *vascular* type it may be possible definitely to trace the lesion to a prolonged and tedious labour, resulting in asphyxia neonatorum. Convulsions may have been present in the first few days of life, and as time passed it was found that there was paralysis and defective growth of one or more limbs, with subsequently mental deficiency. More rarely there may have been a direct injury to the child *in utero*, or an accident to the head in early childhood, whereby a meningeal or cerebral hæmorrhage had been brought about; or the same result may have occurred during a violent paroxysm of whooping-cough. In the *toxic* group of cases the history is generally as follows:—At an age usually between six months and two years the child becomes acutely ill; there is fever, often convulsions and unconsciousness, and other signs indicating that a lesion of the brain or its membranes is present. Such a train of symptoms may supervene during or shortly after an attack of one of the infectious fevers, &c., or they may arise without any such disease; in the former case presumably a localized encephalitis or meningo-encephalitis has been set up; in the latter case it is probable that the infection is a primary one, similar to that described by Strümpell as polio-encephalitis, and analogous to acute anterior poliomyelitis. It is possible also that many cases of so-called "sunstroke" are really of this nature.

Without going into details regarding the pathology of these lesions, it may be said that in all the cases there probably exists a larger or smaller area of brain-tissue in which many of the cells are destroyed; that in addition there may be a secondary overgrowth of neuroglia and a cellular proliferation of the overlying membranes. It is impossible here to discuss more fully the manner in which such a lesion produces amentia, beyond saying that in some cases it may be due to the psychological value of the cells destroyed, and in others to the fact that epilepsy is induced, the amentia then being caused by the severity and frequency of the fits. The fact that so many of these cases are accompanied by epilepsy, and often also by paralysis, leads me to think that this latter is the more common *modus operandi*, the amentia being really an incidental phase of a gradually progressing dementia, although in some cases the lesion certainly results in mental deficiency without either epilepsy or paralysis. Whatever the actual process may be, we may recognise in these cases two distinct forms, firstly, those in which the damage having been done, there is no further spread of the lesion; secondly, those in which a gradual and progressive extension of the process seems to take place, giving rise to frequent epileptiform fits, mental deterioration, and finally dementia. (*See* Plates IX. and X., Figs. 15, 17, and 18.)

It will be readily understood that the prospects of improvement are very different in these two classes; in the former, it is true, a permanent scar of more or less magnitude remains, but judicious and persevering training may do much to improve the mental deficiency resulting therefrom, probably by bringing into play other nerve-cells to take the place of those which have been placed *hors de combat*. Some degree of mental deficiency is, however, almost certain to persist, and the amount of this determines the class in which the patient shall be placed,

PLATE IX.

Secondary Amentia due to Stationary Cerebral Lesion.

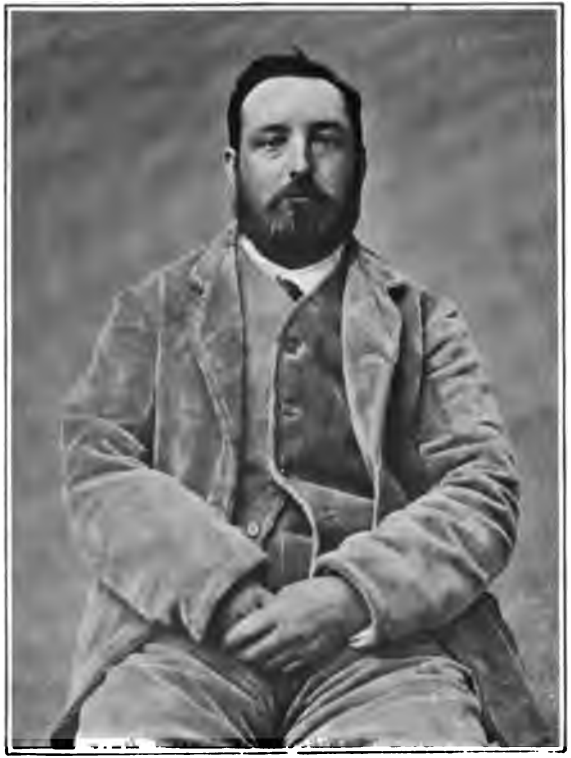

Fig. 15. *Vascular lesion (Asphyxia neonatorum). Mild amentia with paralysis and arrested development of right hand, forearm, and foot. Used to have occasional epileptic fits. Is simple and childish, but can do many odd jobs and shows no signs of dementia.*

Secondary Amentia due to Idiopathic Epilepsy.

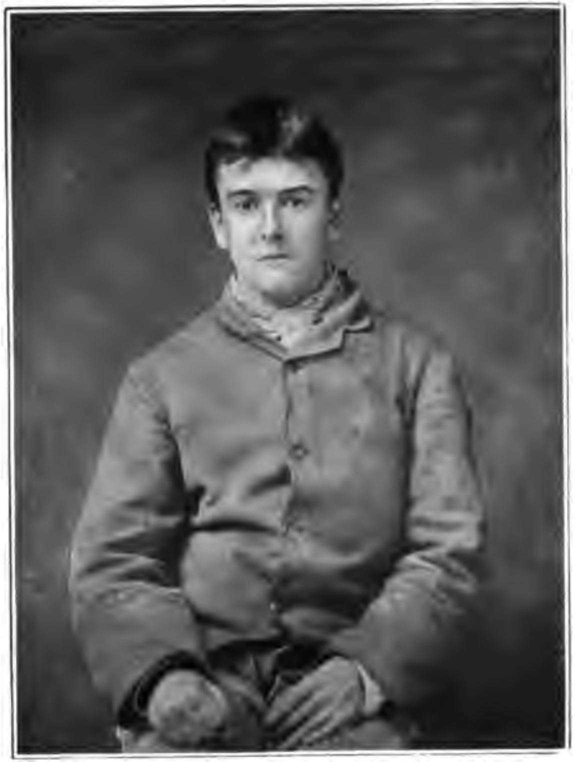

Fig. 16. *Slight morbid heredity present. Epileptic fits since childhood producing a mild degree of amentia, to which dementia is now becoming superadded.*

PLATE X.

Secondary Amentia due to Progressive Cerebral Lesions.

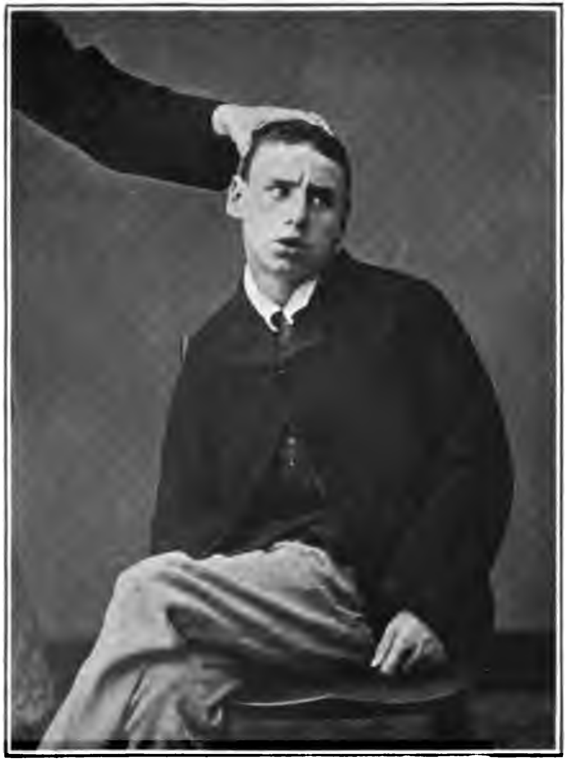

Fig. 17. *Toxic lesion. Encephalitis at age of* 3 *years, followed by amentia with frequent and severe epileptic fits. Gradually became demented and died aged* 17 *years. Post-mortem—atrophy of left cerebral hemisphere with softening in supra-marginal region.*

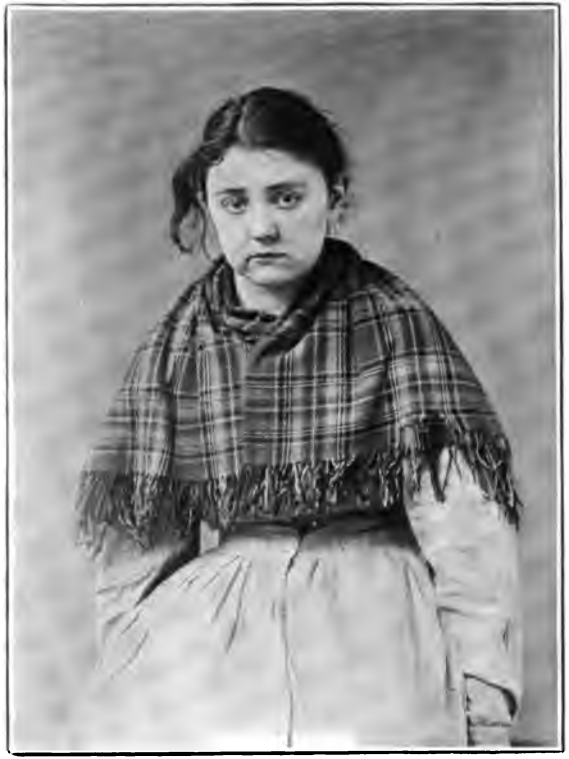

Fig. 18. *Toxic lesion. Slight paresis of right side with epileptic convulsions since one year old. She is now aged* 20 *years, has* 20 *to* 40 *fits monthly, and is becoming demented.*

whether feeble-minded, imbecile, or idiot. When frequent epileptic fits occur, it is but rarely that improvement takes place, the patients gradually losing what few acquirements they have possessed, and becoming dirty, degraded, and finally completely demented.

Dependent upon the particular pathological process we may therefore recognise several clinical varieties, of which the chief are: (1) *vascular* (including traumatic), (2) *inflammatory* (meningo-encephalitis, &c.), (3) *hydrocephalic.* It is unnecessary to say anything further about the first two of these; with regard to amentia *produced by* hydrocephalus, however, although we still know little as to the actual cause of the hydrocephalus and therefore deem it better to consider it *sui generis*, it may be remarked that spontaneous arrest of the disease sometimes takes place, and that after such an event material improvement in the mental condition may result; indeed it has even been asserted that the increased cranial capacity which has been produced may actually lead to greater subsequent development of the cerebral cells, and thus to phenomenal ability. Several notable men with heads of a hydrocephalic shape have been adduced as evidence in support of this theory (?). It occasionally happens that a considerable degree of hydrocephalus may be evident without any obvious mental impairment, but too often, with a gradual increase of fluid, mental deterioration and general helplessness steadily progress until the fatal issue.

It is necessary to emphasise the fact that cases of amentia due solely to one or other of the above causes are by no means common, although the assurances of fond parents might lead one to infer otherwise. They will much more often be found to be contributory or complicating factors of primary amentia, especially of the lower degrees.

"*Paralytic Idiocy*" is frequently spoken of as a specific variety. If it be meant that the amentia is the result of the paralysis the term is absurd, since paralysis, like the mental defect, is merely a symptom of some underlying lesion. If on the other hand the term is meant to include all cases of amentia accompanied by paralysis, its use leads to much unnecessary confusion, since as we have seen almost all the varieties of both primary and secondary amentia may be so complicated.

(B.) *Amentia due to Nutritional Defect.*

The growth of the brain, like that of any other organ, is dependent upon an adequate supply of suitable nourishment, and in those children who, through parental neglect, are systematically deprived of fresh air, sunshine, and proper food, we not uncommonly find a certain amount of mental backwardness. Such, however, is slight, and hardly amounts to defect. We know, however, that one at least of the internal secretions of the ductless glands exercises a most profound influence upon the development and activity, not only of the brain, but of the whole body, and possibly time will show that there are others having a similar influence. I refer to the thyroid gland. In those children in whom this secretion is defective there results that curious condition called *cretinism;* such defect may be the result of disease, or of congenital absence of the gland. As to the ultimate cause we know little, but there does not appear to be any neuropathic diathesis, and the mental condition is purely symptomatic. As is well known, great improvement often follows the administration of the glandular extract, if this treatment be begun sufficiently early. Such cases are not common, and probably do not constitute more than a fraction per cent. of all aments. The degree of mental defect varies very much in different patients, being usually one of feeble-mindedness or mild imbecility, gross idiocy being rare; epilepsy and paralysis are also extremely uncommon. (*Vide* Plate XI., Figs. 19 and 20.)

(C.) *Amentia due to Sensory Defect.*

Amentia resulting from a primary deficiency of the organs of special sense is a rare condition, but a few remarkable, indeed almost classical, examples have been recorded. Whether the cause of the original defect be the result of disease or due to congenital anomaly, the mental condition is entirely secondary, and due to the fact that the child is thereby cut off from communication with the external world, and so deprived of the chief means by which development of the mind can take place. In a few well-known cases this defect has been circumvented by the training of other sensory pathways, at the expense of

PLATE XI.

Secondary Amentia due to Nutritional Defect.

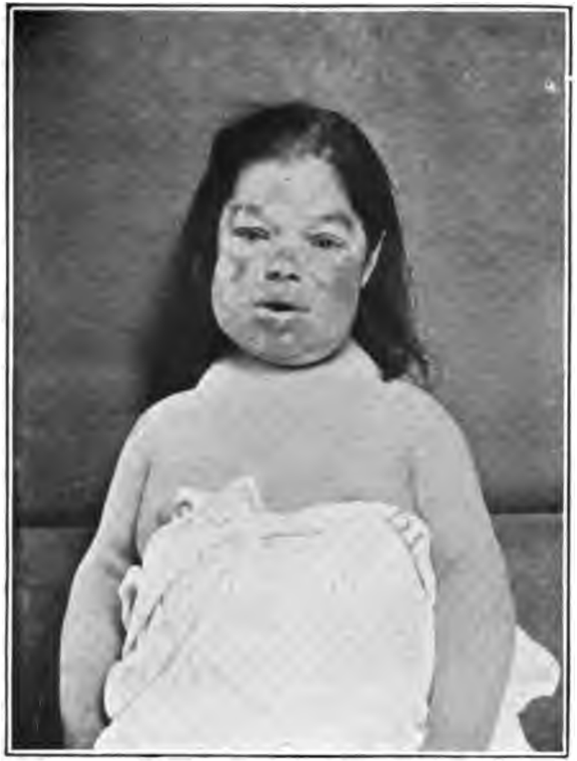

Fig. 19. *Cretin, aged* 39 *years.*

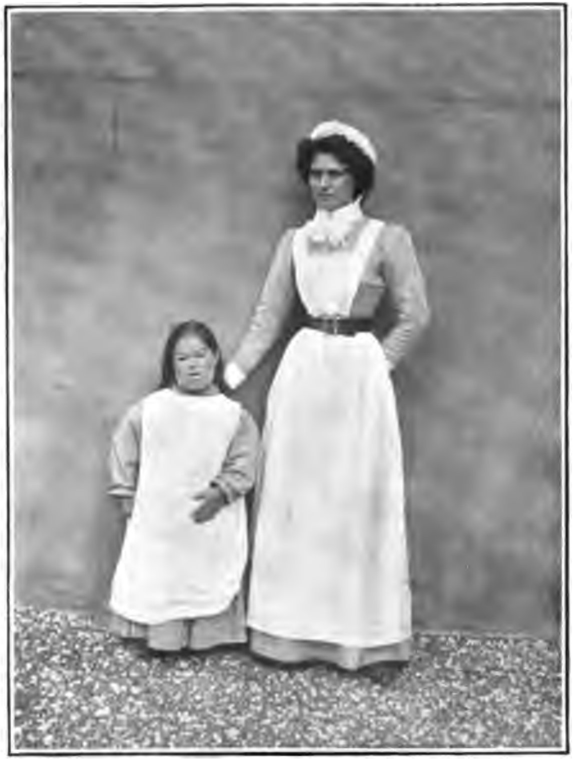

Fig. 20. *The same Cretin, showing relative stature.*

endless patience and perseverance, but with an almost incredible improvement of the intellectual condition. It is important to distinguish between cases like the above and those of primary amentia complicated by some sensory defect, this latter condition being not at all uncommon. The history of the case in the former, or stigmata of degeneracy in the latter, should suffice for an accurate diagnosis.

(D.) *Amentia due to Degenerative Process (as an incidental Phase in the Course of Dementia).*

It is well known that under certain conditions a degeneration of the higher cortical neurones takes place, which is clinically manifested as dementia. The process may be rapid, the patient being brought to a practically mindless condition in a few months, or it may be very gradual, even extending over many years. It is obvious that if this pathological process occurs in early childhood, whilst intellectual development is still far from completion, its first effect will be to put an end to that development, and so to produce a condition of secondary amentia ; its next to rob the child of what acquirements it already had, producing a progressive degradation and ultimate dementia. The amentia then is not only secondary, but is also merely an incidental phase of a progressive dementia, and these cases stand apart from all others hitherto considered. As far as I know there are but three clinical types. The first is the most common and the most important.

(1) *"Eclamptic" and "Epileptic" Amentia.* — We have seen that nearly all the varieties of primary and secondary amentia may be complicated by convulsions; indeed I calculate that these occur in about 40 per cent. of all cases. In the majority of the primary group the fits are due to the presence of ordinary idiopathic epilepsy accompanying the amentia, and it is, perhaps, not surprising that a very considerable portion of those whose brains are imperfectly developed should at the same time suffer in this way. In the minority the convulsions are due to a coëxisting brain-lesion, and in these cases they are often at first Jacksonian in character, but later on become indistinguishable from the ordinary variety. In secondary amentia we have seen that a gross lesion may frequently produce both epilepsy and

amentia. Finally, we come to the present group of cases, in which *idiopathic* epilepsy, ultimately ending in dementia, incidentally gives rise to amentia.

It is therefore of the utmost importance to distinguish between epileptic fits which *accompany*, and those which *produce*, amentia, the two conditions being usually confused in one heterogeneous group of "epileptic idiocy," a term which is really only applicable to the cases we have now to consider.

In a large number of patients the fits have made their first appearance during dentition, and there has either been one or more series of many fits, followed by complete cessation with subsequent mental impairment; or else they have not been so numerous at first, but have continued at intervals for many years. In the former group of cases I think it probable that the convulsions are often really symptomatic of one of the pathological lesions already described, which has escaped recognition; in the latter group, however, they are the first manifestation of actual idiopathic epilepsy, and a neuropathic inheritance will often be found to be present. Sir William Gowers states that in 12 per cent. of cases of epilepsy the convulsions make their first appearance before the age of three years, but many of them date from dentition; although it must be remembered that teething-convulsions are exceedingly common, and are not necessarily epileptic.

Although stigmata frequently occur in these patients, the degree of mental defect is out of all proportion to such indications of degeneracy; and this fact, with the history of the case, will usually render the diagnosis easy. Permanent improvement is practically out of the question, and rapid degeneration usually results. (Plate IX., Fig. 16.)

(2) *Infantile Cerebral Degeneration* (Amaurotic Family Idiocy).—Although Ireland considers this as a "genetous" form of idiocy, and Sachs looks upon it as an example of arrested development, the condition is in reality a purely degenerative one, as is fully shown by more recent knowledge of the clinical and pathological features of the disease, and especially by the work of Kingdon and Russell. It usually begins in the first few months of life with muscular weakness; this is followed by characteristic changes in the optic discs,

ENTIA.

perfect or arrested development.
r, Idiocy.

	CLINICAL VARIETIES.
'angement, and *ɔnes.* :ies :— = ıs :— :lass A. = :ntia.	The chief VARIETIES (dependent upon type of cranial conformation, &c.) are :— (1) *Simple or ordinary.* (2) *Microcephalic.* (3) *Mongolian.* (Also *Scaphocephalic, Negroid, Grecian, and American-Indian, &c.*) The chief SUB-VARIETIES are :— (*a*) Dependent upon gross developmental abnormalities. { Porencephalic. { Sclerotic. (*b*) Dependent upon secondary pathological processes. { Vascular. { Inflammatory. { Hydrocephalic. Epilepsy and paralysis are common particularly in those with gross lesions.
ınes, often with (meningeal or (generally halitis. itis. . or secondary	*Two chief groups occur according as to whether the lesion is stationary or progressive.* The chief VARIETIES (dependent upon pathological cause) are :— (1) *Vascular* (including traumatic). (2) *Inflammatory* (toxic). (3) *Hydrocephalic.* Epilepsy and paralysis are commonly present.
cal neurones due	(1) *Cretinism.* Epilepsy and paralysis rare.
ıind cannot take *ɔs being closed*	(1) *Amentia due to sense deprivation.*
ıeurones often ɔtic changes.	(1) *Epileptic Idiocy, Imbecility, &c.* (2) *Infantile Cerebral Degeneration* (amaurotic family idiocy). (3) *Juvenile General Paralysis.*

the patient gradually becomes almost completely paralysed and demented, and generally dies before the age of two years. In many respects the condition is similar to that of Juvenile General Paralysis, although the onset is earlier and the course is more rapid.

(3) *Juvenile general paralysis* affords a better-known example of an acute degenerative process incidentally giving rise to amentia. The disease is apparently a primary neuronic degeneration, brought about by the poison of syphilis (congenital); it usually begins between the ages of 12 and 15 years, and is completed within four or five years. I have frequently known these patients to be thought suffering from primary amentia, and as such sent to a training establishment. This mistake should not be made, since a little care will show that the process is a progressive one, and that it can be traced to a more or less definite onset, before which the patients were of normal intelligence. The prognosis is absolutely hopeless. In this place it may be mentioned that a patient suffering from primary amentia may of course fall a victim to juvenile general paralysis; indeed, if in addition to the mental defect congenital syphilis be present, there is an extremely strong probability that this will be the termination of the case.

The accompanying table of classification shows the relations existing between the ætiological factors, pathological conditions, and clinical aspects of these different groups and varieties of amentia.

DIAGNOSIS.

There are three questions which we shall be expected to answer when consulted with regard to a child supposed to be mentally defective :—(1.) Is amentia really present? (2.) Can the condition be improved by training, and to what extent? (3.) What is the form of treatment best suited to the child?

The Diagnosis of Amentia.

Mental backwardness does not necessarily indicate imperfect or arrested mental development, but may be the result of a general condition of bodily ill-health, or of neglected education. Many children suffering really from defective nutrition become lethargic and listless mentally, and others may be considerably behind their compeers in acquirements, simply because they have not been taught. Further, there are examples of notably clever men who have been backward in childhood. When, therefore, one of these children is brought to us because suspicions have been aroused that it is not quite "all there," it is necessary to bear these facts in mind. We shall avoid error, and be enabled to arrive at a correct diagnosis not only of the presence of amentia, but also as to whether it is of the primary or secondary form, by paying attention to the following points :—

1. The family history.
2. The presence of stigmata of degeneracy.
3. The physiological development.
4. Indications of abnormal nervous action.
5. Examination of the mental capabilities.

1. *The Family History.*—In the great majority of cases of primary amentia pronounced morbid heredity is present, usually in the form of ancestral insanity or epilepsy, alcoholism, or tuberculosis; whilst in secondary amentia, except when due to epilepsy, such is absent.

2. *Stigmata of Degeneracy.*—In the secondary form these are absent, and the patient is usually well-grown and well-formed, although there may be evidence of some paralytic lesion. In the mildest degrees of primary amentia stigmata are not obtrusive, and may even be wanting altogether, but in the larger number of these cases of medium or pronounced degree the mental imperfection is associated with bodily defect, so that these marks become of the utmost importance from a diagnostic point of view. Care must be taken not to confuse the specific signs of syphilis (which are not necessarily indicative of mental defect) with these stigmata of degeneracy, the chief of which are as follows :—

Cranium: Deviations from the ordinary shape and size, asymmetry, frontal bosses.

Palate: High, narrow, cleft, V-shaped.

Teeth: Irregularities of size, shape, and number; longitudinal and transverse striæ; caries.

Lower jaw: Unusually projecting or receding; asymmetrical.

External ear: Unusually large or small; very projecting; lobules deficient or adherent; imperfect antihelix and helix; supernumerary auricles.

Eyes: Opacity of media; coloboma iridis; heterophthalmos; small and abnormally placed palpebral fissures; epicanthic folds; errors of refraction; strabismus.

Nose: Flattened and depressed, deviation of nasal bones; wide, fleshy nostrils, looking outwards rather than downwards.

Lips: Coarse and thick; hare-lip.

Tongue: Unusually large; fissures, enlarged papillæ.

Skin: Very coarse; localised hairy growths and nævi; excessive development of hair; unpleasant odour.

Hands: General coarseness and clumsiness; irregularities of nails; polydactylism, syndactylism.

Genital Organs: Hypospadias; epispadias; undescended testes.

Limbs: Arms disproportionately long; legs short and bowed.

Body: Generally clumsy and ungainly; stature below average; hands and feet blue and cold; nutrition often defective in spite of good feeding; chilblains common; herniæ.

3. *Physiological Development.*—In secondary amentia the bodily and mental development of the child proceeds in a perfectly normal way, until the latter is suddenly arrested by the onset of one of the acute processes already described. Occasionally also in some cases of primary amentia mental imperfection may not have been noticed before the onset of a similar pathological process, but in the bulk of the cases of the latter variety enquiry or examination will show that the physiological development of the child has fallen markedly behind the average standard. It may therefore be of assistance to give here the chief developmental data:—

In the normal child ability to distinguish between sweets and bitters is present from a few days after birth. Sensibility to painful impressions is present in the early months, although it is then by no means so keen as at a later age. The voice of the parents is usually recognised before the end of the fourth month, and the child begins to notice objects during the sixth month. Two lower central incisors should be cut from the sixth to the ninth month; at the end of a year the child should have six teeth, at the end of two years 16, and by the end of two years and a half a set of 20. The child should begin to say "papa" and "mamma" at the age of twelve months, and by the end of the second year it is able to string together short baby sentences; from this, progress is usually very rapid, the child speedily acquiring the names first of persons and then of objects. Girls talk from one to two months earlier than boys. Voluntary attempts at grasping objects usually occur by the end of the fourth month, a child should be able to stand alone for a short time by the end of the twelfth month, and attempts at walking should be made one month later, and be freely performed by the end of the fifteenth month. The following table gives the average cranial circumferences:—

TABLE OF NORMAL CRANIAL CIRCUMFERENCES.

Age.	End of 1st Month.	2nd Month.	3rd Month.	6th Month.	9th Month.	12th Month.	18th Month.	24th Month.	7th Year.	14th Year.	Adult.
	in.	in.	in.	in.	in.	in.	in.	in.	in.	in.	in.
Males -	14½	15	16	17	17½	18	18½	19	20¾	21¾	22
Females -	14	14½	15½	16½	17	17½	18	18½	20½	21½	21¾

It must be remarked that abnormality of development in any single particular is of little diagnostic value, and especially is this so with regard to the age at which walking is performed and the eruption of the teeth, since these may vary within wide limits even in healthy children. If, however, any marked general backwardness of development is found, which cannot be accounted for by bodily ill-health—especially rickets—and if with this are associated a neuropathic family history and stigmata of degeneracy, amentia is almost certainly present. It may thus be possible to diagnose the condition in the early months of life, even before any examination under the two following headings can be satisfactorily made.

4. *Indications of Abnormal Nervous Action.*—Valuable information may often be obtained by observing the action of the lower neuronic systems, since, as has been well shown by Dr. Warner, there is frequently a correspondence between these

and mental action. But whilst some of these abnormalities of nervous action are certainly indicative of mental defect, others simply denote nervous instability, which may possibly be due to ill-health; and some of these signs may be found in children and adults of even a high degree of ability, almost, indeed, amounting to genius, although in others they are associated with hysteria and potential insanity; care is therefore necessary in their interpretation. The most important of these signs are the following:—Stolidity, vacuity and lack of expression, as well as excessive action in the form of grinning, frowning, and general restlessness denote mental defect, as also do any marked excess or deficiency in the constant small movements which are so characteristic of healthy childhood. Incessant rhythmic movements resembling athetosis are usually the result of a coarse brain-lesion, which may occur in either primary or secondary amentia; whilst on the other hand the sudden, spasmodic twitches of face, shoulders, hands, &c., commonly known as "tricks" or "habits," although indicating a nervous child, do not as a rule denote a defective one. In many cases of amentia the ocular movements are restricted; actual strabismus is often present, and the power of fixation is poor, the latter being probably due to a deficiency of attention. True nystagmus is usually caused by a coarse lesion. Frequently in the defective child volitional power is poor, leading to a shuffling gait and a general listlessness and "lackadaisical" manner; incoördination is also common, resulting in clumsiness in walking and in manipulation, a defective balance and unusual postures. Amongst the abnormal sensory signs of amentia may also be mentioned a general impairment of sensibility to touch, pain and temperature, imperfections of sight, hearing, taste, and smell, and faulty muscular sense. In the merely nervous child, on the contrary, the senses are often of more than average acuteness.

5. *Examination of the Mental Capabilities.*—Conversation affords the best means of forming an opinion as to these. In a few minutes' chat any pronounced mental defect will soon reveal itself; and if the conversation be suitably directed, not only will valuable information as to the psychological action be gained, but the mildest degrees of feeble-mindedness will rarely escape detection. By this means we notice abnor-

malities of speech, or the patient may repeat the question or be utterly unresponsive; by showing him simple pocket objects and asking their names we not only gauge any auditory or ocular defect, but also his power of attention, comparison, and memory; by asking him their uses we obtain a knowledge of his comprehension, his power of description, imagination, and ideation, and in general his degree of judgment and intelligence. Whilst, finally, a few questions put to the friends will acquaint us with his power of control, his moral character, and his general habits.

In this way we shall seldom have much difficulty in saying whether primary or secondary amentia is present; and, from the remarks which have been made in the preceding pages, we shall readily decide which particular variety of these conditions we have to deal with.

PROGNOSIS.

Sixty years ago cases of pronounced amentia were considered to be absolutely and hopelessly beyond any possibility of amelioration. In 1846 Séguin showed that very great improvement might result in certain cases from patient and systematic training, and since that time the education of the mentally defective has made great strides, until to-day it occupies a more or less important position in the social economy of most civilised countries. At the present day, indeed, it is questionable if there is not almost a tendency to over-estimate the educational possibilities, and to think that the machine only needs to be sufficiently elaborate in order that the entering idiot may emerge an individual of ordinary intelligence. Each of these views—the extremely pessimistic and the unduly optimistic—must be avoided, since we now know that there are comparatively few cases so bad that they may not be improved to some extent, and, on the other hand, practically none in which a "complete cure" can be said to take place; there will always remain some slight deficiency which will render competition on an equal footing with the normal population impossible.

To what extent, then, may these cases be improved? Concerning the feeble-minded it is an undoubted fact that under systematic training in suitable classes or homes many of these patients, who would otherwise have swollen the ranks of the loafing and criminal population, acquire steady and methodical habits and a higher ethical standard, and become sufficiently adept and industrious partially, or even entirely, to support themselves under kindly supervision.

Many of the imbeciles, although rarely fit for life outside an institution, can, nevertheless, be trained to occupy themselves with some profitable form of work, such as tailoring, cobbling, shoe-, mat-, and rug-making, wicker, cane, garden, farm, and laundry work, &c.; and, indeed, in a suitable colony they can be made to pay the cost of their maintenance.

A certain percentage of even the lowest class of all—the idiots—although incapable of any useful employment, may, nevertheless, be taught to control their destructive and noisy propensities, to keep themselves clean, and frequently to feed and partially dress themselves, and so to require less attention from more useful members of society. We see, therefore, that education which is suitably adapted to the individual capabilities may do much for these patients, not only in making them more useful to the community and less of a burden to their friends, but also in increasing their happiness.

I would like to take this opportunity of emphasising the absolute necessity for such appropriate treatment, and of strongly condemning the too frequent custom of completely neglecting and hiding away these little patients in holes and corners. Such will inevitably intensify the original defect, besides leading to the development of habits which will not only be a source of endless worry and sorrow to the relatives, but which may become an actual menace to society.

It is now necessary to refer to the prognosis which must be given in any particular case. Generally speaking, it may be said that cases of primary amentia are more amenable to treatment than are those of the secondary form; in other words that, despite their appearance, more can as a rule be done for those with repulsive features, misshapen bodies, and stigmata of degeneracy than for those who are well-formed and well-favoured. The reason for this is that in the former we have to do with a general neuronic imperfection, which may be brought to a higher state of development by training, whilst in the latter actual destruction of brain-tissue has taken place.

This general statement must, however, be modified to some extent, since considerable improvement often occurs in cases of secondary amentia in which the lesion has been but small, and has become stationary, provided that treatment be begun early enough ; also by appropriate methods where it is the result of athyroidism or of special sense-deprivation, as deafness, blindness, &c. On the other hand some of the cases of gross primary idiocy are hopeless from the very beginning, but with these qualifications the above general statement holds good.

In those cases in which the amentia is but an incidental phase of a progressive degeneration, the prognosis is of the worst, and as a rule the presence of mental instability, insanity, paralysis, and above all severe epilepsy, render the prospects of permanent improvement very small and makes education scarcely worth attempting.

TREATMENT.

It is beyond the scope of this article to describe the many special educational methods which have been devised for the training of these patients, and these can only be carried out by skilled teachers under the supervision of a medical expert. But although the practitioner need not concern himself with these details he will be expected to indicate to what extent they are applicable, and more particularly to advise as to the choice of a suitable institution.

In the earliest years of life treatment will almost of necessity have to be carried out at home, and the physician should impress upon the mother that even at this age much may be accomplished if she will only be sufficiently patient and persevering. It is of the utmost importance to inculcate habits of personal cleanliness ; if this can be secured, the child may be gradually taught to dress himself at least partially, and subsequently to amuse himself with a few bricks, whilst later on the various appliances of the kindergarten may be made use of. All these have a definite educational value, and must preface any attempts at more direct intellectual training. It will also be the duty of the physician to pay particular attention to the general bodily condition, to see that any remediable disease is treated, *e.g.*, adenoids, enlarged tonsils, errors of refraction, &c., and to take care that the matters of food, clothing, and exercise are suitably provided for.

Mental training should follow physiological and psychological lines, and should embrace (1) the development of the sensory channels, both peripheral and central, by means of objects of different size, texture, and weight, such as size—peg- and form-boards, &c. (tactile); music, &c. (auditory); form-boards, coloured cubes, and the matching of variously-coloured ribbons, &c. (visual); (2) the coördination of motor action by suitable calisthenics; and lastly, the education of the higher faculties by means of pictures, object lessons, &c. Imitation of the teacher's movements, hand- and finger-exercises, and for higher-grade patients practice with graduated weights held in the palm, as recommended by Dr. Warner, are of the utmost service in affording simultaneous instruction in all these directions.[1]

But, however favourable may be the conditions of home life, they can never compare with the advantages to be obtained in a special institution where the very companionship of children of similar age counts for not a little, and the interests of the child will be best consulted by getting it into a suitable training-school as early as it is possible to do so. The selection of such an institution will necessarily be governed by the degree and variety of amentia, and by the social position and means of the parents.

For those who can afford to pay a sum ranging from 120*l.* to 250*l.* yearly, there will be no difficulty in finding a private home fulfilling the requirements.[2] For those who cannot meet such an expense it may be of service to know that patients are received at Earlswood Asylum,[3] without limit as to age, at terms ranging upwards from 65 guineas annually; also at the Royal Albert Asylum,[4] at a similar rate, and that the training in either of these institutions is excellent. In addition, non-pauper patients may be elected into Earlswood Asylum without charge, by the votes of subscribers, but this is often a tedious process requiring much canvassing; if part payment of a sum not less than 15 guineas annually is made, however, less votes are required. The Royal Albert Asylum also receives patients belonging to one of the seven northern counties, at the reduced charge of 25 guineas annually. For the feeble-minded, the National Association[1] has a number of excellent homes at which the payments range from five to twelve shillings weekly.

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[1] For further information upon these points the reader may consult Dr. Shuttleworth's book on *Mentally Deficient Children* with advantage.

[2] A list of private homes will be found in Wright's *Medical Annual.*

[3] Address—Medical Superintendent, Earlswood Asylum, Redhill, Surrey.

[4] Address—Secretary, Royal Albert Asylum, Lancaster.

Unfortunately for those of the pauper class the provision is at present far from adequate, and considerable difficulty may be experienced in obtaining the requisite education. In London and a few of the large provincial towns advantage has been taken of the Defective Children's Act to form special classes, and in these places the requirements of the feeble-minded are well met; it is, however, to be regretted that the Act did not make it compulsory upon the educational authority to provide such classes, instead of leaving it to their option.

For pauper imbeciles and idiots training is provided at Darenth Schools in Kent, and at the Middlesex county and a few other county asylums, whilst both of these classes may be received under certificates in any of the county lunatic asylums. I would, however, warn the practitioner against the too hasty certification and incarceration of these patients in the latter institutions, since the majority of them are not adapted for, and do not profess to be able to carry out, any educational training. If the child be very troublesome, or should actually become insane, as may happen, these asylums must be had recourse to; but if there seems any prospect of amelioration, no efforts should be spared to try first the effect of education in a special home set apart for that purpose.

For the photographs illustrating this article, many of which were specially taken, I desire to express my thanks to Dr. McDowall, of Morpeth Asylum (Figs. 5, 6, 7, 8, 9); Dr. Moody, of Cane Hill Asylum (Figs. 15, 16, 17, 18); Dr. Robert Jones, of Claybury Asylum (Figs. 1, 2, 3, 4), and Dr. C. A. Marsh, of Darenth Asylum (Figs. 10, 11, 12, 13, 14, 19, 20).

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[1] **Address—National Association for Promoting the Welfare of the Feeble-minded, 53, Victoria Street, London, S.W.**

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SOME IMPRESSIONS OF THE SANATORIUM TREATMENT BY A "HEALTHY PATIENT."

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[With Charts I. to III.]

HAVING had the opportunity of visiting several sanatoria both in England and abroad, it seemed to me that a short holiday might profitably be spent in actually undergoing the open-air treatment, and looking at the matter from the patient's point of view. This I was enabled to do through the kindness of my friends, Dr. M. S. and Dr. F., who received me as a patient into their admirably managed sanatorium in April of the present year. It may be of interest briefly to record some of my impressions.

The method of treatment followed in this institution is strictly on the lines of that of Nordrach, and may be summarised as follows :—

At 7 a.m. the patients rise. Each takes a shower-bath of a medium temperature, a cold douche being prohibited in cold weather, lest the deep inspirations produced might rupture recent adhesions. The patient then wraps himself in blankets, dries himself slowly to avoid undue exertion, and then slowly dresses.

8 a.m. Breakfast.

At 9 a.m. the patients commence their walks, returning to their rooms by 12 o'clock.

12 to 1 p.m. This hour, preceding the midday meal, is devoted to resting on the reclining-chair in the bedroom.

1 p.m. Dinner.

At 2 p.m. the patients start for their afternoon walk, returning by about 5 p.m.

6 to 7 p.m. Rest-hour spent on the *chaise longue* in the bedroom.

7 p.m. Supper.

After this meal half an hour is spent in conversation or in listening to music (piano or violin-playing, not singing), if the Sanatorium can boast of a performer.

Shortly before 9 o'clock the patients return to their rooms and retire to bed, the electric light being extinguished at 9.30.

Such is the daily routine for those patients who are non-febrile and convalescent, and this was the life which I followed during my stay in the Sanatorium, walking with the various patients, and being dieted in a similar manner.

My experience, I may say at once, proved a most enjoyable one. The Sanatorium contained some 20 patients, of whom 6 were in a somewhat acute stage, and remained in bed, while the remaining 14 were non-febrile, and took walks of various length. With these patients—those most suitable for Sanatorium treatment—I was continually in contact, and rarely has it been my good fortune to meet more cheery and pleasant companions. Though some had been ill and under treatment for a considerable time (two certainly for more than a year and one for two years), no word of complaint was ever uttered, and each one by carefully obeying orders was fully determined to regain his health.

Among the many points which interested me, perhaps what struck me most was the rapidity with which one became accustomed to the open-air treatment. Having always suffered greatly from the cold, and the weather being very wintry, I expected to suffer somewhat severely. But though it froze hard (15 degrees of frost being registered on one occasion), I very rapidly became acclimatised, and delighted in the stimulating effect of the cold air blowing through the room. In this respect my own experience is the usual one, and only very rarely does it happen that a patient (generally one who is liable to rheumatism) is obliged to abandon the treatment.

As the result of this perpetual life in the fresh air, the appetite became greatly stimulated, and the liberal diet ordained was taken without much difficulty. As a result my weight increased rapidly, a gain of 3 lbs. being noted in less than a week. Neither at this Sanatorium, nor, indeed, at Nordrach at the present time, though the diet is plenteous and nourishing, is any attempt made at "forced feeding" as the term was originally understood, and in both Sanatoria I have seen patients voluntarily ask for second helps.

Another good result which seemed in my case to follow directly from the open-air life, was increased ability to sleep.

In London it would have been difficult, if not impossible, to go to bed at 10 o'clock and fall asleep at once, and to sleep on until called in the morning. And yet this was my experience at the Sanatorium, and it is no uncommon one.

A frequent charge brought against the Sanatorium treatment is that its monotony must prove terribly trying. No doubt after some time the daily round may become irksome, but my experience proved to me that, for the Nordrach system at least, the monotony had been greatly exaggerated. Given pleasant companions and pretty scenery (and in these respects I was most fortunate) the six hours of exercise pass easily and pleasantly, while the quiet resting hours before dinner and supper form an agreeable change, in which reading of any kind, intellectual or otherwise, or even sleep, may be indulged in. It is, indeed, a common complaint among patients—and there is some truth in it—that the day is so carefully and fully mapped out that they have no time for doing anything, even writing home. Whether as much can be said for the strict Liegehalle system, where much more time is spent in lying out in the open air, and less in taking exercise, is open to question. It is obviously easier to supervise in this way a larger number of patients, especially when these are drawn from the lower classes, but it would seem that to the Englishman at least the system must be more irksome and wearisome, while it cannot but tend to the putting on of fat, rather than to strengthening the muscular system and improving the general vitality. Even here, however, the monotony may be somewhat diminished by permitting to a limited extent open-air games, such as croquet or garden golf, or now and then organising miniature shooting competitions, concessions which are now advantageously made at most sanatoria.

Another point which interested me much was the very considerable amount of muscular exercise performed by the patients in their slow walks. A healthy person is apt to think, when he sees the patients, like the ploughman of old, slowly wending their way at the regulation rate of about two miles an hour (this pace being chosen so as to prevent dyspnœa, and therefore the overtaxing of the lungs) that but little active exercise is being taken. Let him, however, make the attempt himself, and he will see the fallacy of his idea. Each step in very slow walking requires sustained muscular exertion, for the progress is so slow that, were the foot not actually held up, it would fall to the ground before the body is sufficiently moved forwards to be ready for this event. As a result, four or five miles of such walking prove even to the man in perfect health a distinctly tiring performance, and the wisdom of ordering an hour's rest in the Sanatorium on the return home before the next meal becomes apparent. The value of the latter in increasing the appetite is also easily proved to any one who has noticed and compared the difference between eating immediately after a walk, or only after such an interval of rest.

There is one drawback, however, which this slow walking possesses, and it is one which might be easily corrected. It is that so often the patients continually fix their gaze upon the ground, thus allowing the shoulders to fall forwards and compress the chest. An elastic band passing from shoulder to shoulder would easily and advantageously correct this tendency.

It is an aphorism that the walks in the immediate neighbourhood of a sanatorium should be as much as possible uphill, so that the harder portion of the allotted task may be performed when the patient is fresh. But there is another reason for this, in that sometimes too long a rest is taken before the return journey is commenced, and the latter is therefore somewhat hurried. If this be so the patient is less likely to get out of breath, and the lungs to be over-exerted, if he be going down hill, than if he is breasting an incline on his return to the Sanatorium. In this respect, and also in the variety of the walks and their varied shelter, the Sanatorium at L. is most fortunately placed.

It is thus evident that even to a healthy, active, "experimental patient," a sanatorium in a suitable neighbourhood, ably conducted on the Nordrach system, is by no means the dull, monotonous place which people may sometimes imagine. Given agreeable companions—and but few phthisical patients would be excluded from this class—the time passes pleasantly, while the exercise taken, besides strengthening the tissues and improving the appetite, gives a sense of "something accomplished, something done," which is so eminently satisfactory to the Englishman. The resting hours may also well

CHART 1.

Showing the rectal exercise-rise after a 3 hours' slow walk of about 4 miles.

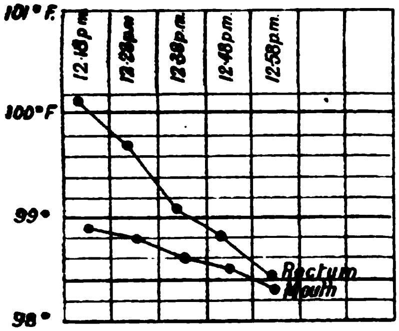


CHART 2.

Showing the rectal exercise-rise after a slow walk of about 3 hours.

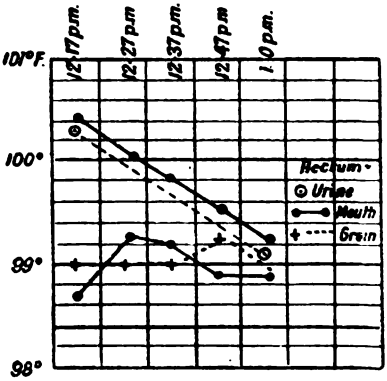
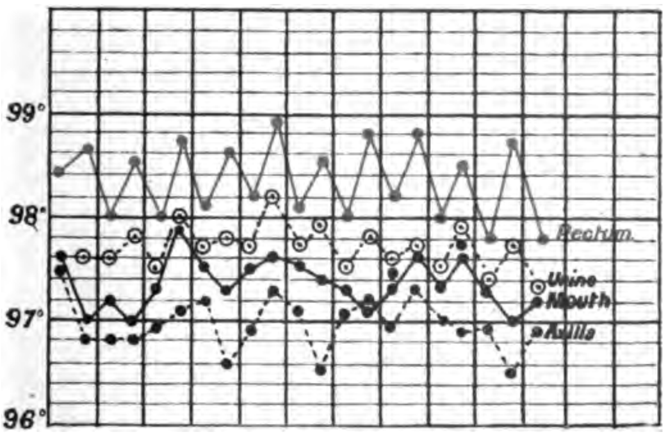


CHART 3.

Showing want of correspondence between the mouth and axillary temperatures (when taken for five minutes) as compared with those of the rectum.



be spent not in merely vegetating and novel-reading, but, as the patient recovers his health, in more serious study, which will be useful to him hereafter.

Before concluding, a few words may be devoted to the question of temperatures, a constantly recurring theme of interest at all sanatoria.

At this institution which I visited, as at Nordrach, the temperatures of the patients are always taken in the rectum. The method is not ideal from an æsthetic point of view, and its use can only be justified if important data are obtained in this way, which cannot be arrived at in any other. At present, opinion is divided upon this point, the followers of Dr. Walther strongly advising its use, while others, like Dr. Burton-Fanning (who in a recent important publication[1] has done much to advance our knowledge on this subject), believe that the temperature may be satisfactorily taken in the mouth provided due regard be paid to certain conditions set forth in his paper. As these, however, in not a few cases necessitate the keeping of the thermometer in the mouth for considerable periods of time, fifteen minutes or more, before the true temperature is recorded, a practical difficulty is introduced. In the great majority of cases it is impossible to ask the patient to retain the thermometer in the mouth, groin, or axilla for more than five minutes, and what, therefore, we now require to know is whether it is safe to rely upon the temperatures registered in this manner in these situations or whether recourse must be had to rectal temperatures.

The following charts, from observations made upon myself, are of interest in this connection. The temperatures were taken for five minutes in each case with "half-minute thermometers" verified at Kew.

Charts I. and II. (especially the latter), taken immediately after three hours of slow walking, confirm Dr. Burton-Fanning's conclusions that the normal exercise-rise can only be observed with certainty in the rectum, temperatures in the mouth and groin (and I may add also in the axilla) failing signally to show it. In the case of the former this is owing doubtless to the cooling of the oral cavity from breathing with the mouth open during walking, though as regards the groin the reason is not so obvious.

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[1] *The Lancet*, March 28, 1903.

The following figures show a similar result, the temperatures having been taken immediately after returning from a sharp walk of five miles :—

Rectum - - - - -	101·6
Urine (small quantity) - - -	101·0
Mouth - - - - -	99·2

If, therefore, it is important to know the extent of the exercise-rise—and in some cases it is possible that this is of value—it is clear that we cannot rely upon the mouth or groin, and that the rectum must be our guide.

Chart III. shows the record of my temperature taken night and morning for a period of ten days after my return home. The morning temperatures were registered between 7 and 8 a.m. on first waking, the evening ones at about midnight, after sitting quietly reading or writing for three or four hours, thus absolutely excluding the effect of exercise. The readings were in all cases taken after five minutes. An examination of the chart shows that in the morning the temperature of the mouth may be from ·5 to ·9 degree below that of the rectum, while in the evening the difference may amount to 1·7 degrees. The axillary readings are even less satisfactory.

The closest approximation, in my own case, to the rectal temperature is given by that of the urine (obtained by holding the thermometer in the stream), the latter registering from ·4 to 1·1 degrees below the former. For practical purposes, however, this method of taking the temperature is not to be recommended, seeing that accurate results depend both upon the thermometer registering with great rapidity, and also upon an adequate supply of urine being forthcoming, which is not always the case.

The above observations are few in number, and refer only to my own case, but they are not dissimilar to certain others which have recently been taken for me at the Brompton Hospital, and they certainly tend to support the opinion that in sanatorium work, where the exercise of the patient must depend upon the absence of fever, and where, therefore, accuracy of observation is all-important, the temperature should be taken in the rectum.

HEADACHE.

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The ever-increasing demands of advancing civilisation and the accompanying stress of competitive effort bring in their train new disease-problems to occupy the attention and stimulate the interest of all who are actively employed in the field of medicine, whether in the department of laboratory research, or in that of clinical practice. In no corner of this immense territory of work is there more active development than in that concerned with disorders of the nervous system; in none have the results of laborious investigation been rewarded with more substantial additions to the sum-total of our knowledge of disease; nor is there in any other such opportunity for the cultivation of diagnostic refinement. It must unfortunately be confessed that therapeutic efficiency has conspicuously failed to keep pace with the strides of increasing clinical accuracy. The evolution of the human race is proceeding at a rate unknown in the earlier centuries of man's development and is inevitably creating a multiplicity of new demands upon the originating powers of the mind and upon the resources of the nervous mechanism, with the result that we have to face an acquired instability of neurons which is responsible for many new maladies, as well as for greater frequency and increased intensity of others that have been recognised and handed down through successive generations of men. Nervous exhaustion was unknown until comparatively recent times, and the ancient history of medicine is eloquently silent on the causes which produce it or the symptoms by which it may be recognised. The altered circumstances of modern life have produced increased cares and anxieties, greater hurry, more assiduous attention to work, and a growing taste for the luxurious gratification of sensual pleasures; all of which encourage a neglect of the due relationship between vital income and expenditure.

The organic and functional disturbances of health which result from the increased wear and tear of this more rapid existence are innumerable, and though they differ from one another so widely that their full description would involve a consideration of the whole gamut of disease, most of them are accompanied by one symptom which, from its very prevalence, has attracted less attention than the misery it causes well entitles it to. This symptom is Headache, and whilst it may be impossible to encompass the wide ramifications of its relationships to disease in any attempt at description, yet, from its very omnipresence, it would seem worthy of a more definite individualisation than medical literature has so far accorded it. The fact of its being a mere symptom of a wide variety of disorders is surely no good reason why it should not be treated as an entity! Diarrhœa, convulsions, anæmia, and many other conditions that might be named are, though mere symptoms, accorded descriptive importance, and yet there are few text-books in which headache is to be found as the heading of a chapter, and only a small number of treatises wholly devoted to its exposition. The subject is of obvious importance, and is one which always taxes the experience of the physician and calls into play his best powers of observation and deduction in order to elucidate its nature and indicate the remedies most likely to effect its amelioration or cure. In some cases the cause is apparent and capable of removal by local or constitutional treatment; in others no adequate explanation can be found in the condition of the patient's health and we are driven to seek a solution of the problem in the realms of speculation. It is quite rational to suppose that perverted nutrition or functional disturbance may produce molecular changes in the delicate structure of the nervous system just as effectually as, though perhaps less objectively than, an injury. Pain is the expression of nerve-disturbance; and, if our ignorance too often prevents our finding an explanatory cause, it exists none the less, could we but penetrate deeply enough to unravel the secrets which at present lie buried in darkness and which we fatuously try to make ourselves believe are comfortably settled by being labelled with the high-sounding title of Neurosis.

By the term headache is meant pain in the head, the precise seat of which may be either the structures covering the calvarium—the muscles, skin or fasciæ; the skull bones, or the mucous lining of the various cavities which they contain; or the cerebral membranes. The substance of the brain itself is, in its normal state, incapable of feeling pain, but may possibly become acutely sensitive under the influence of organic disease. On the other hand, it is not improbable that the blood-vessels, the walls of which are supplied with nerves, may be sensitive to painful influences, and many observers from the time of Galen have held them responsible for that form of headache which seems to proceed from the very depths of the brain itself.

ANATOMY.

In order fully to understand the varieties of headache, it is necessary to refer shortly to a few leading anatomical facts.

The head is supported behind by a mass of muscles which, during the upright position, are never at rest, and which are therefore liable to become painful under the influence of fatigue.

The aponeurotic expansion of the occipito-frontalis muscle which extends over the whole of the upper part of the cranium is closely united to the skin, but is divided from the bone below by a loose areolar tissue which allows it to move freely over the skull. It is perforated with holes for the passage of vessels and nerves, and, as the structure is unyielding, it is conceivable that circulatory disturbances of all kinds may readily cause tenderness from pressure and other changes. The temporal fascia, which covers the temporal muscle on either side, is stronger than the epicranial aponeurosis. It consists of two layers which are fixed to the edges of the upper border of the zygomatic arch, and between them there lies some fatty tissue with a small branch of the superficial temporal artery and a twig of the orbital branch of the superior orbital nerve.

The pericranium is loosely connected with the bone except at the sutures, where it is firmly adherent to and continuous with the dura mater. It readily, therefore, becomes implicated in diseases of either the bone or the dura.

The vascular supply of the scalp is derived from the internal and external carotid arteries; the supra-orbital and frontal branches come from the former, whilst the temporal, occipital and posterior auricular branches are derived from the latter. The veins correspond in their course and distribution with the arteries; those accompanying the branches of the internal carotid artery empty into the facial vein, the others open into the jugular veins; they also communicate by small emissary branches with the intra-cranial sinuses, and with the veins occupying the diploë of the cranial bones. The lymphatics of the posterior part of the scalp pass down to glands situated in the sub-occipital region; those from the lateral aspect terminate in glands, situated beneath the parotid fascia and over the insertion of the sterno-mastoid muscle; and those from the anterior aspect join the lymphatics of the face.

The nervous supply is derived from the three trunks of the fifth cranial nerve over the whole half of the head anterior to a vertical section through the two external auditory meatuses; the posterior aspect is supplied by branches of the first and second cervical nerves, and by an offset of the facial or seventh cranial nerve behind the ear.

The dura mater consists of two layers; the outer being closely adherent to the bone and continuous through the sutures and fissures with the pericranium. It provides sheaths to the various cranial nerves, and projects inwards three great processes—the falx cerebri, the falx cerebelli, and the tentorium cerebelli.

The pia mater is closely applied to the surface of the brain and penetrates into its interior at the transverse fissures, where it forms the velum interpositum and the choroid plexuses. The arachnoid occupies the space between the dura mater and pia mater. It forms, like other serous membranes, a sac which contains a lubricating fluid. The subarachnoid space between the arachnoid and pia mater communicates with the great cerebro-spinal cavity through an opening in the roof of the fourth ventricle.

Within the brain the blood-supply is derived from the vertebral and internal carotid arteries. The vertebrals pass from the subclavian trunks through the foramen magnum into the skull, and blend with one another to form the basilar artery which lies on the pons, at the upper border of which it terminates by dividing into two branches—the posterior cerebral arteries; these anastomose freely with the anterior cerebral branches of the internal carotid artery in the circle of Willis. The middle cerebral branch of the internal carotid enters the fissure of Sylvius and ramifies over the outer side of the hemisphere. The quantity of blood within the cranium amounts to fully one-fifth of the whole blood of the body. It is subject to considerable variation, but uniformity of pressure is provided for by compensatory change in the amount of fluid contained in the ventricles and other cavities of the subarachnoid spaces and in the perivascular sheaths surrounding the cerebral blood-vessels. These all communicate with one another and contain a watery fluid, the amount of which is in inverse proportion to the amount of blood contained in the vessels. Where cerebrospinal fluid is in excess vascularity is slight, but where it is diminished turgescence is increased.

CAUSATION.

It is impossible, within the limits of a short article, to make any attempt at a complete ætiology of such an extensive subject as headache, but the following factors have a general bearing upon the question and are important to keep in mind in the investigation of all cases.

Heredity.—The tendency to headache may run through many generations, but it is only a secondary result either of some congenital defect or of a family diathesis.

Age.—In infancy and old age headaches are uncommon. In childhood, brain-tumours or middle-ear disease; and during school life, uncorrected refractive errors, overpressure of work, and unhygienic class-rooms are frequent causes. At puberty, headaches often occur in association with sexual development; and at about the age of 40 presbyopia may be responsible for them. Persistent headache after middle life should always arouse a suspicion of interstitial nephritis.

Sex.—Headaches occur from four to six times more frequently in women than in men; probably because of the greater delicacy and instability of the female nervous system, of their more emotional temperament, and of the demands made upon their reserves of energy by menstruation, pregnancy, and lactation. During the menopause, and sometimes for years afterwards, headaches of a peculiarly severe type are often experienced, and in some women they are an invariable accompaniment of each menstrual period.

Personal Circumstances.—Irregular habits of life are to be blamed for a large proportion of the headaches constantly met with. Excess of food, no less than excess of alcohol, may produce them. Sudden and extreme exertion may beget them, but, on the other hand, so may a life of luxury and idleness. Fatigue, the result of protracted mental or physical effort, or the consequence of prolonged exposure to noise and bustle or to strain on the attention, often finds its first expression in headache. Powerful impressions on the organs of special sense and profound emotion, whether of joy or sorrow, are also exciting causes. It may result from any sudden effort, such as coughing, sneezing, or immoderate laughter, all of which probably produce some passing congestion of the cerebral blood-supply. In women it is sometimes attributable to the way in which they dress their hair. When the hair is tightly drawn back, the roots are dragged upon and the nerves of the scalp are liable to be thus irritated.

Atmospheric Conditions.—Excessive heat or cold, a low barometric pressure, and that electrical state of the air which accompanies a thunderstorm, are each and all capable of causing headache. Lunar influences are also still credited with the power of producing it. The direct rays of a strong sun are an universally-recognised cause, and in sunstroke headache is always a prominent symptom. Impurity of air, such as is found in an overcrowded room, is another common antecedent. A cold, biting north-east wind cannot be tolerated by some people because of the certain headache which follows exposure to it.

Local Disease.—Headache may be induced by a variety of disorders situated either outside or within the cranial cavity. It may also ensue upon traumatic lesions. Of causes situated externally, inflammatory conditions of the scalp or cranial bones, and rheumatic or gouty affections of the muscles or fasciæ, are among the most common. Disorders of the teeth, too, are frequent sources of faceache and pain elsewhere in the head. Dental headache is more frequently unilateral than bilateral, generally involves the front part of the head, and may be confined to a circumscribed area such as the supra-orbital region. It is often met with in association with disease of the upper molars. Of intracranial disorders, tumours and meningitis are the most common causes of headache.

Disorders of the Special Sense Organs.—The eye is the special-sense organ most frequently associated with headache, which may be produced either by organic disease of the globe, such as glaucoma, iritis or conjunctivitis, or by eye-strain from refractive error or imperfect accommodation. As regards the ear, disease in any part of it may cause not only earache, but also reflected pains in various regions of the head. The most usual cause of earache is otitis media, but it is not unfrequently induced by the presence of hardened cerumen in the external ear, or by caries or some other abnormal state of the teeth. It may also accompany disease of the tonsils, pharynx, or tongue, and it is sometimes met with independently of any obvious cause. The pain may radiate from the tympanum or mastoid region to almost the whole of the corresponding side of the head, but is sometimes entirely confined to a temporal or occipital distribution.

The nose, together with its accessory chambers, and the pharynx are, when diseased, fertile sources of headache. It is common knowledge that an ordinary catarrh is frequently accompanied by severe throbbing frontal pain. The nasal cavity and its communicating sinuses are lined with one continuous mucous membrane, so that disease originating in any one part is liable to spread by extension. Headache may be set up either by accumulation of waste products in the brain and meninges from obstruction of the nasal lymphatics, or from hypertrophic changes in the mucous membrane of one or other of the accessory sinuses, with consequent absorption of the air in the cavity involved and a collateral hyperæmia either in the neighbourhood or further away in the meninges ; or from interference with nasal respiration and with the proper aëration of the blood. The situation of the pain in headache from nasal causes is usually just over the eyes, and it frequently involves the bridge of the nose ; that which ensues from disease of the pharynx is generally occipital.

Other Disorders.—Headache is an accompaniment of a vast number of constitutional and other diseases. It may be stated broadly concerning them, that they cause headache either by the structural changes they produce in the blood, or by the toxines which are elaborated as the result of their existence, or by a reflex influence which they exert on the normal equilibrium of the serous and vascular fluids within the brain or upon the delicate structure of the nervous tissue itself.

DESCRIPTION.

In dealing with the subject of headache it is obviously impracticable to describe in detail the various disorders with which it is associated, or even those of which it forms the leading feature. The object aimed at is rather to pick out this element of headache from the various settings in which it is found, and to endeavour to eliminate from its complex associations and manifold manifestations such broad and distinguishing features as may serve to differentiate one variety from another, with a view more particularly to direct special attention to the details of treatment by which it may be hoped most successfully to combat it. No one form of management and no single therapeutic method can possibly avail against a condition of pain which owns such a multiplicity of causes, though the legions of headache-cures which are advertised, and for which marvellous virtues are claimed, tend to make suffering humanity and a too credulous profession alike liable to be deceived into a belief in the possibility of a one-and-only panacea. Any system of classification must necessarily be arbitrary and incomplete, but for a well-ordered general conception of the subject it is requisite to adopt some plan of division whereby one kind of headache may be sharply contrasted with another. Its distinctive features may thus afford a clue to the lines upon which treatment ought to run, and also provide useful data for guiding the differential diagnosis of the diseases upon which it depends. For practical purposes the following arrangement will be found to comprise most of the varieties which possess distinctive features and are of daily occurrence.

1. *Organic Headache.*—This form of headache is produced by intracranial changes affecting either the meninges or the

brain. Among the more common causes which are responsible for it may be mentioned meningitis, tumour, abscess, thrombosis and hæmorrhage. The pain varies in intensity, and is influenced by the rapidity with which the lesion develops and the extent to which the membranes are involved. It is usually continuous and abiding, but may be paroxysmal or even intermittent. It is worse at night than during the day, and is always increased by stooping or exertion. It interferes with and often entirely prevents sleep. Its distribution is usually frontal, but where the cerebellum is the seat of disorder its situation is occipital. It may also be occipital when the cause is an aneurysm of the basilar artery, whilst in some cases of hæmorrhage it is temporal. When the lesion is cortical there may be localised tenderness of the scalp over an area corresponding with its position in the brain. Vomiting unrelated to gastric trouble or food-taking, optic neuritis, vertigo, constipation, and irregularity of the pulse are important collateral symptoms. Other phenomena, such as paralysis, convulsive movements, disordered sensation, or intellectual aberration may arise and enable a more or less accurate estimate of the localisation of the lesion to be arrived at. In cases which are progressive, the patient is often ultimately seized with convulsions or dies comatose. The nature of the lesion must be inferred from the history and progress of the case, the age of the patient, and the effect of treatment. Syphilitic arteritis may give rise to symptoms almost indistinguishable from chronic meningitis or cerebral tumour, but the diagnosis can often be guided aright by an admitted history of specific infection or by the discovery of scars or other external evidence of its previous manifestations. On examination of the eyes, such confirmatory evidence is at times forthcoming in the existence of adhesions from an old iritis, or in the presence of disseminated choroiditis or of optic atrophy.

In syphilitic headache the pain is paroxysmal. It may come on at a certain hour of the night, and only continue until a certain hour in the morning. If it moderates during the day, it is characterised by nocturnal exacerbations, so that the subjects of it are unable to sleep. It may be circumscribed and confined to one spot, but more frequently it is diffused and deep-seated. The occurrence of an epileptiform seizure is an indication that the lesion is advancing towards the cortex and the cerebral membranes.

In cerebral abscess the course of the disease is acute, and there are increase of temperature, rigors, and other evidences of the existence of pus, together with a history of injury or the presence of an infecting focus.

In meningitis, the headache is diffused over the skull and is associated with fever, retraction of the head, delirium, and photophobia. The pain is frequently paroxysmal, causing the patient at intervals to give vent to a sharp cry, and the muscles at the back of the neck are stiff and tender. In thrombosis, if the superior longitudinal sinus is plugged, there may be epistaxis, œdema of the forehead, and exophthalmos. When the cavernous sinus is involved, there is œdema of the eyelids, and subsequently of the entire face on the affected side, accompanied by varying degrees of paralysis of the oculo-motor nerves. When the tentorial sinus is obstructed, the œdema is situated behind the ear, and the jugular vein on that side is often less full than its fellow; and since the commonest cause of the condition is suppurative otitis, there is generally an accompaniment of severe earache. In hæmorrhage, the initial headache may be violent and limited to one or other parietal or temporal region; it is accompanied by confusion of thought, and quickly followed by unconsciousness or convulsions; there is paralysis corresponding with the extent and situation of the lesion, but hemiplegic in distribution in the large majority of cases because of the frequency with which the ruptured vessel is situated near the internal capsule.

2. *Nervous Headache.*—This form of headache is also known as Migraine or Hemicrania. It is usually unilateral and is always associated with vaso-motor phenomena. It occurs more frequently in women than in men, and is especially liable to happen during menstruation. It is often hereditary, and is almost as frequent in childhood as in adult life, but it disappears in old age. It is closely connected with gout and rheumatism, and is generally accompanied by high arterial tension. The attacks are paroxysmal; set in at varying intervals of days, weeks, or years; and are excited by over-exertion, mental excitement, acute indigestion, impure air, or eye-strain. The pain is more common on the left than on

the right side of the head, and generally begins in the early morning either immediately on waking or soon after. It is usually preceded by prodromal disturbances, such as vertigo, languor, tinnitus, excessive yawning, transient hemianopia, spots before the eyes, "fortification patterns," &c.; these being accompanied by mental depression, coldness of the extremities, and chilliness. At first the pain is localised to a spot in the temple, from which it spreads to the parietal and supra-orbital regions, and either remains limited to one side of the head or gradually spreads till the opposite temple, or even the whole cranium, may become implicated. It is continuous, and goes on increasing in intensity until, after a period varying from one to six hours or sometimes longer, the height of the paroxysm is reached, and the nausea by which the pain was accompanied is replaced by retching or vomiting, which often serves to afford relief. There is always extreme sensitiveness to noise or light, and the slightest movement notably increases both the pain and the gastric uneasiness. Finally, a troubled sleep ensues, from which the patient awakes free of pain, but languid and worn out. There is a kind of headache known as *Clavus*, in which intense pain, as though a nail were being driven forcibly into the head, is felt over a limited spot, most frequently in the supra-orbital or temporal region. It may probably be more correctly classified with migraine than with any other form of headache. From time immemorial it has been regarded as a hysterical manifestation, but whereas it is often accompanied by nausea or vomiting and vaso-motor manifestations, it may safely be looked upon as dependent upon something more deeply seated and complex than mere functional derangement, especially in view of the admitted fact that it is, in a large majority of cases, associated with chlorosis.

3. *Neuralgic Headache.*—Though neuralgia may be experienced in any of the nerves supplying the head, it is most common in the trigeminal or fifth cranial nerve. This form of headache is popularly spoken of as tic douloureux. It is rare in children and most frequent in middle life, or at a later period when the tissues are beginning to undergo degenerative changes. It is then often very intractable. Its most frequent cause is exposure to cold and damp, but it may also be produced by the irritation of a decayed tooth, by antral disease,

or by the pressure of a morbid or inflammatory growth near one of the bony canals traversed by the branches of the nerve. It is predisposed to by all conditions of exhaustion or of depressed general health. The pain is deep-seated and of a stabbing, boring, or burning character. It sets in suddenly and may involve any of the three divisions of the nerve. It is always confined to one side of the face and emanates at first in short acute paroxysmal darts which rapidly increase in violence and duration, until they may become so intense as to cause the patient to scream with the agony he suffers. When thus severe, it is accompanied by spasmodic contractions of the facial muscles and vaso-motor phenomena, such as flushing, local sweating, lachrymation and salivation. Tender spots along the course of the nerve are characteristic, and are usually situated at the site of the emergence of the branches of nerve from the bone. When the ophthalmic division is affected, the tenderness to pressure is most marked at the supra-orbital notch or at the inner angle of the orbit; when the superior maxillary division is attacked, it is most pronounced at the site of emergence of the infra-orbital nerve or along the upper gum; and when the third division suffers, the tender points are to be found in front of the ear or at the seat of exit of the inferior dental nerve. The hair is liable to become white over the situation of pain ; the skin may become thickened and pigmented or herpetic; and the portion of tongue within the area of distribution of the affected nerve may become furred from overgrowth of epithelium.

When the posterior branches of the first four pairs of spinal nerves are affected, headache will be experienced in the occipital region, whence the pain may spread over the back and top of the head, and, in some cases, even reach to the forehead. It is frequently accompanied by vertigo, tinnitus, and some mental confusion, and is liable, on that account, to be mistaken for commencing organic disease of the brain. Tender points are found where the great occipital nerve becomes superficial in the posterior triangle of the neck and over the parietal eminence. This variety of neuralgic headache is often bilateral, and the pain is continuous but subject to periodic exacerbations.

4. *Toxæmic Headache.*—In this form of headache, which is the most frequent of all, the pain is attributable to the action

on the nervous elements in the brain of blood which has become altered in its constitution by the addition to its volume of toxines. The toxæmic condition may be induced either by poisons introduced from without or by poisons formed within the body itself. Thus certain drugs, such as iron, quinine, and opium ; certain articles of diet, either because they are directly poisonous or because they are indirectly so on account of the indigestion they cause ; alcohol when taken in more than physiological amount ; and tobacco when indulged in excessively, may all be responsible for it. From whichever of these causes it proceeds, the importance of personal susceptibility must always be borne in mind. One man, dyspeptic all his life, may never have known what headache meant ; while another, on the slightest upset of his digestion, experiences headache which unfits him for anything. To one person alcohol or tobacco seems innocuous even in liberal allowance, whilst to another the most moderate indulgence in either muddles his head and incapacitates him temporarily for any form of mental work. It is common knowledge that many patients with interstitial nephritis in which the system is exposed to constant risk of blood-poisoning, do not suffer at all from head-troubles, whereas others are never free of them from the time the kidneys begin to undergo structural change. Headache accompanies all acute fevers and inflammatory disorders, and is a feature of diagnostic importance in some, such as influenza and enteric fever. It occurs as one of the phenomena of gout and rheumatism, is constant in cases of Bright's disease when the secreting tissues of the kidneys are so impaired that they no longer rid the blood sufficiently of its waste, and is frequent in association with various forms of hepatic disorder. Breathing the impure air of crowded or badly-ventilated rooms is another common cause. In short, in all cases of blood-poisoning or contamination, from whatever source arising, headache has to be considered as one of the invariable symptoms.

The pain is of a dull, heavy character, generally occupies the forehead and temples, is often associated with flushing of the face and injection of the conjunctivæ, and is accompanied by physical exhaustion and nervous prostration. Its duration and degree vary according to the exciting cause ; and though of itself it seldom serves to justify a diagnosis, the headache sometimes presents features which, when read into the text of the general condition, help to clear up an otherwise obscure symptom-complex.

In influenza, for instance, it is frontal in distribution, but is strikingly intense over the globes of the eyes, which are often so tender that they dare not be touched.

In enteric fever it is frequently the earliest symptom, and may be continuously persistent for a week or more before the development of the disease produces other phenomena distinctive enough to explain its nature. In mild cases with little disturbance of temperature, the presence for days of headache, slight in the morning, but becoming more severe as the day goes on, should always arouse suspicion that there is more behind the symptoms than a mere febricula.

The headache produced by drugs disappears on their withdrawal, or may in many cases be obviated by their admixture with suitable correctives. Quinine can often be borne when combined with hydrobromic acid, iron when prescribed along with salicylate of soda, and opium when associated with alkalies.

Dietetic headache has its origin in imperfect digestion of food in the stomach or duodenum and may be caused either by direct irritation of the pneumogastric and sympathetic nerve-supply or by absorption of toxæmic products. It rarely comes on immediately after food is taken, since some time must elapse before the ingested materials become irritating or toxic from fermentative or putrefactive processes. The toxines thus formed are, under normal circumstances, destroyed by the liver, but if this organ becomes congested and deficient in biliary secretion, they rapidly accumulate in large amounts and enter the blood-stream. Young people of both sexes are liable to this variety of headache, but among adults men commit greater excesses in eating and drinking than women, and consequently suffer more frequently. The pain comes on in the morning after excess of food or alcohol the night before, or it may succeed a full or indigestible meal taken at any other time of day, if this is eaten hurriedly and followed by active exertion. The pain is vertical as well as frontal, and is apt to be pulsating and accompanied by nausea and vertigo, especially on stooping. It usually disappears after a night's sleep; or relief is obtained by active vomiting, or by the occurrence of colicky pains and diarrhœa by which the irritating matters are expelled from the intestines. During the continuance of the headache there is an unpleasant sense of discomfort at the epigastrium, the urine is thick from urates and scanty, the tongue is furred, and the mental condition is one of depression; any attempt at intellectual work brings about an increase of the pain and confusion of ideas, and the complexion gradually becomes muddy or icteric. So great is the sympathy between the stomach and the brain that a headache which was at first simply bilious may lead on to a true migraine.

The influence of the gouty or rheumatic diathesis in exciting severe headache is universally recognised. In a patient who has suffered from one or more attacks of articular gout, the sudden onset of severe frontal headache and giddiness—especially if attended by changes in the urinary and alvine evacuations which indicate disorder in the functions of digestion and assimilation, by a high-tension pulse and accentuation of the second cardiac sound over the aortic cartilage, and associated with a history of preceding irascibility—should always suggest the possibility that retrocedent gout may be the true explanation. The rheumatic headache is of quite a different type; it happens to a patient who has either previously suffered, or is at the moment suffering, from rheumatism in some other part of the body. It affects the epicranial aponeurosis and the occipito-frontalis muscle, especially at its tendinous attachment over the brow and occiput, and is often induced by exposure to cold or damp. The pain is severe, but superficial, and the scalp is tender to touch. It is worst in the evening and grows less towards morning, but is at all times liable to short paroxysmal increase. It is neither so severe, nor does it render the patient so powerless, as the pain associated with gout. It is, moreover, of much less serious consequence, and is not accompanied by the risk of delirium and other cerebral symptoms which occasionally supervene in the gouty condition.

In renal disease headache is either an evidence of uræmia or it is a direct result of high arterial tension. The headache of uræmia occupies the entire forehead, is dull though severe, and is often associated with an uncomfortable sensation of fulness, ringing in the ears, dimness of sight, or actual blindness, and a tendency to delirious excitement and subsequent drowsiness. There is frequently vomiting and diarrhœa, both of which are to be looked upon as eliminative efforts, and muscular twitchings or convulsions may supervene. Confirmation of the nature of this form of headache is afforded by an examination of the urine, and by the frequent presence of albuminuric retinitis. When the cause of the uræmia is contracting kidney the arterial tension is increased, the cardiac action is tumultuous, there is a loudly-accentuated second sound, and the pulse is sometimes slow and often irregular. Oppression in the chest with breathlessness on exertion are common accompaniments.

5. *Ocular Headache.*—The headache of eye-strain is produced by abnormalities in the ocular muscles; most frequently by weakness of the external or internal recti or by astigmatic errors of refraction. Mild degrees of astigmatism often become manifest only when presbyopia begins. Minor ocular errors are more likely to cause headache than those which are pronounced, because any great defect of accommodation leads to the abandonment of all attempts to call it into play, the patient seeing as best he can without it. It is still uncertain whether muscular asthenopia *per se* can cause true migraine, though some observers believe it to be possible. But putting this contentious point on one side, ocular abnormalities are responsible for a form of headache which differs from all others in the following particulars. The pain may be referred to any part of the head, but is most commonly complained of in the occipital region. After prolonged use of the eyes in reading or writing, there is blurring of vision, pain on movement of the globes, hyperæmia of the conjunctivæ, and a tendency to congestion of the lids. These symptoms rapidly subside under the influence of rest of the eyes. The pain of glaucoma is more local, and is usually felt about the orbit or in the eyeball. At first it is often unilateral, and is then apt to lead to a false diagnosis of neuralgia. Ophthalmoscopic and retinoscopic examination of the eye is necessary to establish the diagnosis whenever the symptoms above narrated suggest an intra-ocular cause as being the explanation of the pain.

6. *Neurasthenic Headache.*—The headache which is associated with nervous exhaustion is probably immediately due to an obscure form of toxæmia, but it possesses characteristics of its own which are sufficiently distinctive to entitle it to separate mention. It is frequently associated with intercurrent exacerbations which are characteristically migrainous, the two varieties being closely allied ætiologically. The pain may be superficial or deep ; it is always occipital in situation, and is frequently associated with a sense of constriction as though the head were encased in an iron sheath—the *casque neurasthénique* of the French. It gives rise to such hyperæsthesia of the scalp that the sufferer dare not brush out or even touch the hair ; and it is painfully increased by light, noise, or any other form of extraneous disturbance. It is always accompanied by vasomotor phenomena, emotional instability, impossibility of mental effort, and anorexia. The presence of the other general phenomena of neurasthenia — disturbed digestion, muscular tremor, mental introspection, polyuria, loss of bodily weight, irregularity of bowels, menstrual insufficiency, insomnia, and persistent languor—supply confirmatory evidence of the nature of the headache, but especially the existence of an active knee-jerk—the elicitation of which causes a responsive contraction of the muscles of the trunk as well as of those of the leg, so that the whole body is jerked off its seat—and a complete absence of plantar reflex. This form of headache continues as long as a person who is overworked persists in fatiguing himself, and rapidly disappears when sufficient and complete rest is taken. It is the headache of the "present century," and is undoubtedly caused by the stress of life and the increasing demands of ambition for long-sustained effort without the recuperating influence of adequate leisure and repose.

7. *Hæmic Headache.*—Headache may be associated either with plethora or anæmia. Plethoric headache is dependent upon fulness of the vessels of the brain, and occurs mostly in persons of a full habit of body. It is also met with at the commencement of pyrexial disorders, in threatening apoplexy, in certain forms of valvular disease of the heart, in sudden menstrual suppression, and after an epileptic seizure. The pain is dull and continuous, usually extends from the forehead to the occiput, and is accompanied by heaviness and stupor. On

turning suddenly, uncomfortable vertigo may be experienced, and the pain is increased by stooping. When the headache is long-continued the patient is apt to become hypochondriacal, sleep is disturbed by unpleasant dreams and often by copious perspirations; the bowels are confined, and the urine is high-coloured and loaded with lithates. The face is bloated and flushed, the eyes are lustreless and heavy, and the conjunctivæ are injected. The pain is increased by emotional disturbance or mental excitement. Persons who are subject to this form of headache usually live freely and prefer sedentary habits to active exertion. It is essentially the headache of alcoholic excess.

Anæmia, from whatever cause it arises, is a very common cause of headache. The pain is of a persistent and often acute type, is most frequently vertical, but is in some cases frontal and in others occipital. It is apt to assume the neuralgic type, and is then confined to one side of the head or limited to the supra-orbital region. It is relieved by the recumbent or stooping posture, and is accompanied by noises in the ears, dizziness, flashes of light before the eyes, throbbing in the head, a tendency to fainting, palpitation of the heart, and pallor of the skin and lips. The pulse is slow and feeble, the bowels are confined and the extremities cold. Mental depression is invariable and is apt to induce a desire for alcohol, which affords temporary relief by dilating the vessels of the brain and stimulating the force of the heart's contractions.

(To be continued.)

THE TREATMENT OF PARAPLEGIA IN TUBERCULOSIS OF THE SPINE.

SOME SUGGESTIONS AND A REVIEW OF RECENT WORK.

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[With Plates XII. to XVI.]

WHEN paraplegia supervenes in the course of spinal tuberculosis the seat of the disease is generally at or above the mid-dorsal region.

The plan of treatment most commonly adopted in this form of paraplegia is by rest either in the recumbent or the prone position; and this plan is not uncommonly successful. In some cases, however, the paraplegia is of rapid onset, with supervening myelitis; in such cases operation must be resorted to without delay; in other cases rest in the most favourable conditions, although patiently tried for one or more years, fails even to relieve the patient's sufferings and at most prolongs a dying life.

As an alternative to rest laminectomy has been recommended as a routine measure, but it has not had any wide acceptance; and both on pathological and on clinical grounds I have not been able to subscribe to this view, save in so far as it applies to certain cases; *e.g.*, those of sudden onset, and those rare instances in which the arches of the vertebræ are involved in the disease, and a few other exceptional conditions. Among the objections to laminectomy are the following:—By the removal of the sound arches the spine is further weakened at the seat of the disease; when tubercular matter has been evacuated by the route opened up by laminectomy, the tissues round the whole circumference of the cord may become infiltrated by tubercular invasion, and permanent damage to the spinal cord (whether by constriction or by myelitis) may ensue.

Another resource consists in forcible correction of the angular deformity in the spine. Several instances are on record in which paraplegia, which has been present at the time when this operation has been done, has begun to improve immediately afterwards. Now, although my own experience has not led me to favour forcible correction of deformity in tubercular spondylitis, as a proper or necessary part of the treatment of the condition when uncomplicated with paraplegia, I have held that it would be justifiable in a case of paraplegia that had resisted treatment by a year's rest in bed in either the prone or the supine position, combined with proper fixation of the spine and proper dietetic, medical, and hygienic measures. This view I have abandoned in favour of a less hazardous proceeding, viz., opening up a passage to the front of the vertebral column by a lateral operation.

It will be of interest, then, to pass in review recent work bearing on this subject, but first it is necessary to obtain a clear idea of the clinical conditions to which apply the various therapeutic measures about to be considered. The following group of cases may be recognised :—

1. Cases in which the paraplegia (chiefly motor) comes on gradually and gradually disappears when the patient is put to bed.
2. Cases in which the paraplegia (chiefly motor) persists in spite of rest in bed with extension of the spine.
3. Cases in which the paraplegia develops rapidly, is both motor and sensory, and, if not soon relieved, passes into myelitis with bed-sores, &c.
4. Aberrant cases ; *e.g.*, where pressure is due to bone and not, as is usual, to the formation of solid or liquid tubercular matter at the front of the spinal cord.

As examples of the results to be obtained by laminectomy, the cases published by W. Arbuthnot Lane in 1890–1 may be taken as examples :—

Case I.—Patient aged 32. Apex of projection at tenth dorsal vertebra. On removing laminæ a large mass of granulation-tissue, resembling tubercular synovial membrane, protruded ; it contained in its centre about ½ drachm of pus. The whole growth was removed, together with a small process which extended forwards to the right of the dura-matral sheath. Recovery (before the patient was allowed to be up) was complete.—(*Lancet*, July 5, 1890.)

Case II.—A youth, aged 16, wore Sayre's Jacket for three months. It was not replaced. The patient got about with difficulty, and paraplegia gradually developed. Sensation was impaired. The fourth, fifth, and sixth dorsal spines and laminæ were removed. The cord was found to be splayed out over the firm wall of a tense abscess-cavity which came into relation with its anterior surface. The abscess was as large as a Tangerine orange. A drainage-tube was left in for thirty-six hours. Rapid improvement occurred at first; then subsequent return of paraplegia. A second operation showed that the cord was surrounded by abundance of caseous material, which had extended into the muscles and parts in the vicinity for a considerable distance. As much as possible of this material was removed, but with little benefit, the patient leaving hospital little better than he entered it.—(*British Medical Journal*, June 6, 1891.)

Case III.—A female, aged 21; disease of four years' standing. Recovery followed the first operation. Five months later complete paraplegia again developed. The cord was exposed and found to be splayed out over a large abscess-cavity. This was evacuated and scraped, and drained with injections of iodoform and glycerine for several weeks.

Case IV.—Boy, 7½ years. The cord was found to be compressed between the body of fifth dorsal vertebra and adjoining laminæ. The immediate result was good. When reported, the child was waiting for a poroplastic jacket.—(*British Medical Journal*, April 20, 1889.)

Up to June 6, 1891, Lane had operated by laminectomy on eight patients, all bad subjects. One relapsed; one showed no improvement; five were permanently relieved; one died from rectal polypus.

Detailed comment on this series of cases may be reserved till after an alternative method of treatment, namely, by a lateral operation, has been considered.

C. B. Keetley (1890) performed "lateral trephining" for paraplegia in spinal disease. In the case of a boy aged 14, after having performed laminectomy, Keetley at the same time trephined the body of the vertebra corresponding to the angle of the curvature by perforating the transverse process and pedicle (on the right side, in a direction from without, inwards, and forwards). "I used some rather large centre-bits made specially for the purpose, and worked by an ordinary drill, enlarging the canal thus bored by means of a gouge and sharp spoon. I did not find any tuberculous focus, or granulations, or pus." There was no immediate improvement, and although the patient ultimately recovered Keetley did not attribute this to the operation.[1]

Ménard (*Revue d'Orthopédie*, Nov. 1, 1894) recommended "costo-transversectomy" as a substitute for laminectomy in cases of spinal tuberculosis complicated by paraplegia. Ménard was first led to perform this operation by two considerations:—First, in a case in which laminectomy was done a tubercular cavity was accidentally opened, and the patient began to improve immediately; second, in another case in which he had done laminectomy 2½ years previously without any benefit being obtained, he subsequently explored the sides and front of the diseased vertebræ after removing several transverse processes; immediate and progressive improvement followed.

The experience that first led me to perform costo-transversectomy was as follows:—

Case A.—A lady, aged 45, was sent to me by Mr. William Armstrong (Buxton) in August, 1891. She complained of pain between the shoulders and inability to stoop. These symptoms had been present for eight months; four months after a fall from a ladder, which was followed by pain in the epigastrium. On examination of the back the spinous process of the eighth dorsal vertebra was seen to be slightly prominent. There was complete rigidity of the spine in the dorsal region, and a girdle-pain extending round the body from the eighth dorsal spine forwards and downwards to a little above the navel. A skiagraph (Fig. 1) showed a dark area on each side of the spine in the whole of the dorsal region; the lateral extent of this area was greatest opposite the right side of the eighth dorsal vertebra. The patient, who was somewhat delicate and subject to severe migraine, was put to bed, the spine being fixed by an antero-posterior splint. In spite of rest, nutritious diet, &c., symptoms of paraplegia supervened early in October. The patient complained of a heavy feeling in the legs. In the course of ten days the paraplegia rapidly became complete, both sensory and motor: the bladder and sphincter ani were also paralysed. After a consultation with Sir William Broadbent and Mr. William Armstrong, I performed laminectomy on October 23. When exposed, the spinal cord in its sheath bulged backwards, and on passing an aneurysm-needle round the right side of the cord a

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[1] Keetley: *Orthopædic Surgery.*

PLATE XII.

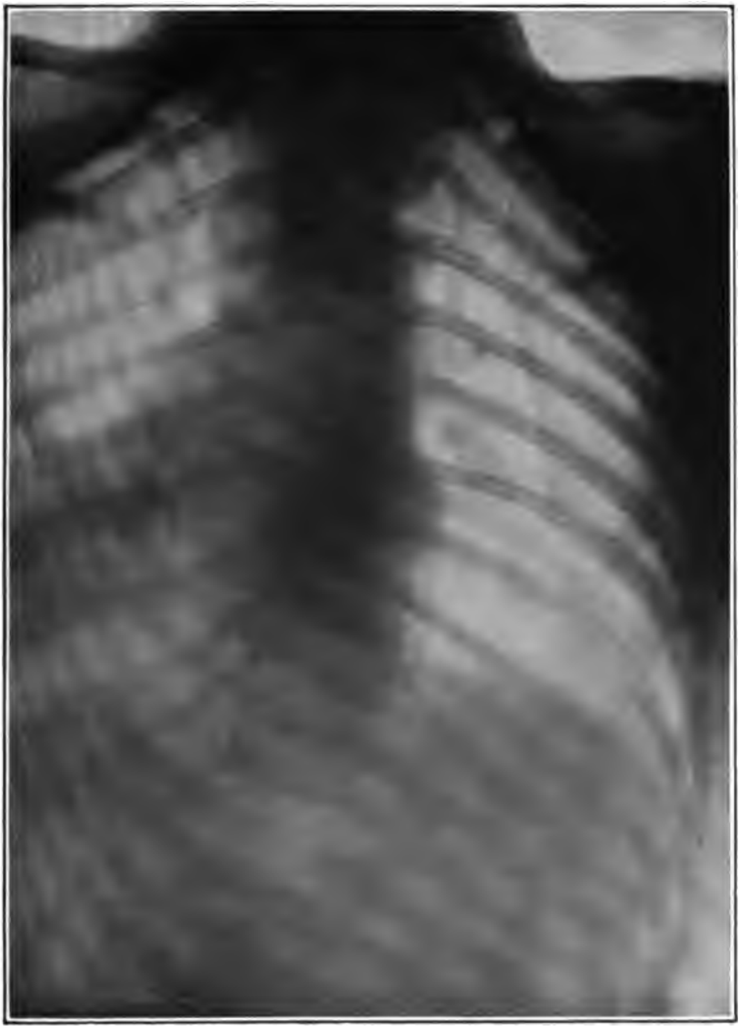

Fig. 1. *Skiagraph of the spine of a patient, aged 45, showing diffuse thickening of the whole of the dorsal spine: there was an abscess between the 7th and 8th dorsal vertebræ.*

small abscess-cavity was entered, giving exit to about a tablespoonful of pus. As the pus escaped, the cord sank into its place. The abscess-cavity was found to be bounded in front by the remains of the bodies of the seventh and eighth dorsal vertebræ; thus the condition was that somewhat rare one—a posterior spinal abscess. The immediate result of the operation was satisfactory; some sensation and bladder-power began to return. I felt, however, that the recurrence of suppuration in this case was almost inevitable, and that, if it occurred, it would follow the track by which the abscess had been evacuated, and might then lead to a condition similar to that recorded above in Case II. of Mr. A. Lane's. To meet this possibility, a week after the laminectomy, I removed the right transverse process of the eighth dorsal vertebra and the neck of the corresponding rib, thus providing an easy exit for any pus that might form at the site of the abscess. After the transverse process and the portion of rib had been removed, I was very strongly impressed with the fact that *had this second operation (costo-transversectomy) been performed in the first place, the laminectomy would have been altogether unnecessary.* After this second operation the patient had a severe attack of migraine, which, combined with the effect of the anæsthetic (ether), caused a critical period, during which I found the coöperation of Mr. William Armstrong of infinite service. Apart from this, the patient did well. Both wounds healed soundly and the improvement in sensation was maintained. The motor functions of the cord, except the slowly-increasing power in the bladder and some action in the sphincter ani, still remained in abeyance. Five months after the operation the value of the costo-transversectomy was proved. An abscess formed under the lateral scar; it was easily opened under aseptic conditions, and was kept aseptic under a dressing of bicyanide gauze. A small sinus remained until early in 1903, when it closed, and the scar became depressed; and *pari passu* with this depression of the scar came a most gratifying return of power in the lower limbs, which has steadily increased from February to the present time (September). The patient will soon be allowed to walk with crutches and a spinal support. That the cord has undergone some organic change is evidenced by slight ankle-clonus and the presence of Babinski's sign on both sides.

In another case I performed costo-transversectomy directly for the relief of paraplegia with a completely satisfactory result. This patient I showed before the Clinical Society of London (October 24, 1902) nine months after the operation, and she has been uninterruptedly under my observation up to the present time. She is now in good general health; and, supported by a light apparatus, she has been allowed to walk and run about for two years past. The deformity in the spine is very slight, and she has safely weathered attacks of measles and scarlet fever. The case [1] is deserving of record in some detail:—

Case B.—Eliz. R., aged 4, was admitted to the North-West London Hospital on May 11, 1901: she was a delicate, thin child, with well-marked beading of the ribs. She had a distinct angular projection of which the fifth dorsal spine formed the apex. Both lower extremities were extremely weak, but the patient when lying in bed could just draw them up, on being requested to do so. The knee-jerks were increased, and there was an extension of the great toe on stroking the sole of the foot (Babinski's sign). Sensation was not impaired.

There was but little tenderness over the seat of deformity, though at night the child would complain of pain there and at the front of the sternum. There was also little wasting of the lower limbs. The temperature was sub-normal, and save for occasional rises to normal remained so until after operation.

History.—The history obtained from the parents was that six months before her admission the patient's legs became suddenly paralysed. After a time they recovered somewhat, but again became weak. The family history was good with the exception that the patient's grandfather died of phthisis.

Immediate Treatment.—Patient was put to bed close to a window facing westward and allowed as much fresh air as possible. Cod-liver oil and iron and a liberal diet were also given.

On July 3 patient was still able to move her legs a little, although they had considerably wasted during the time that she had been in hospital. She soon ceased to complain of pain, slept well and had a fair appetite, but she remained thin and weak.

After she had been in hospital seven weeks, the wasting of the lower extremities was more marked, and the knee-jerks became very faint; the patient's general condition still showed

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[1] This record is based on the notes of Mr. G. W. M. Pritchett, then senior resident at the hospital.

PLATE XIII.

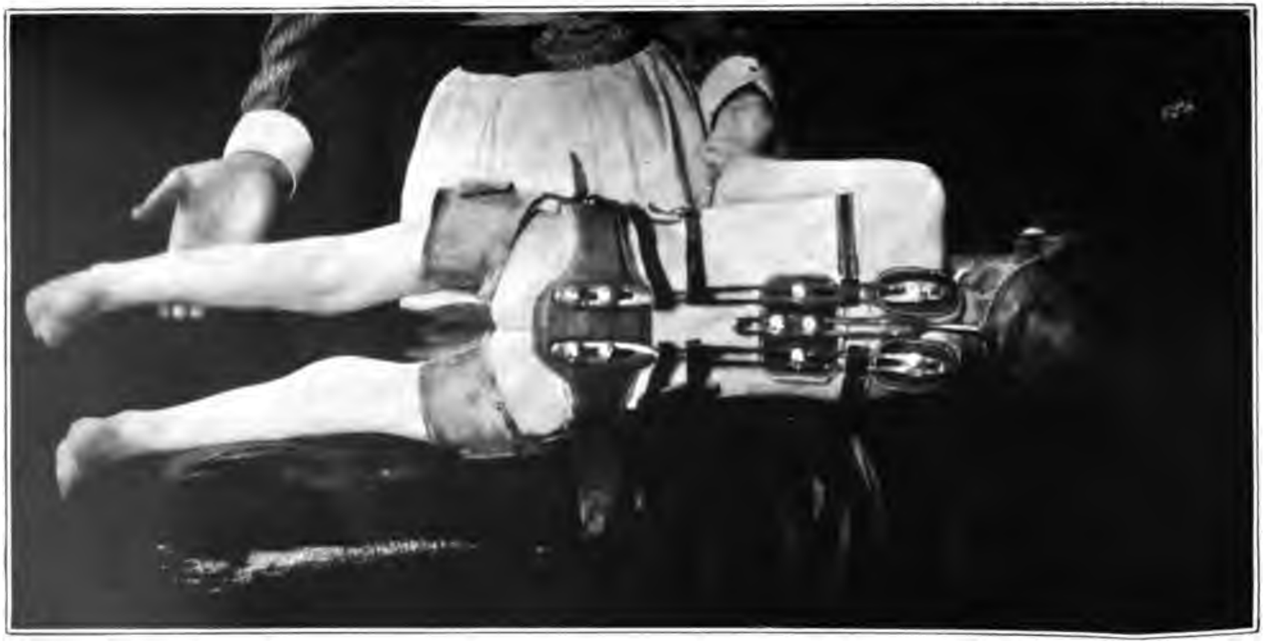

Fig. 2. *Modified Chance's splint applied to a child with high dorsal caries and motor paraplegia.*

PLATE XIV.

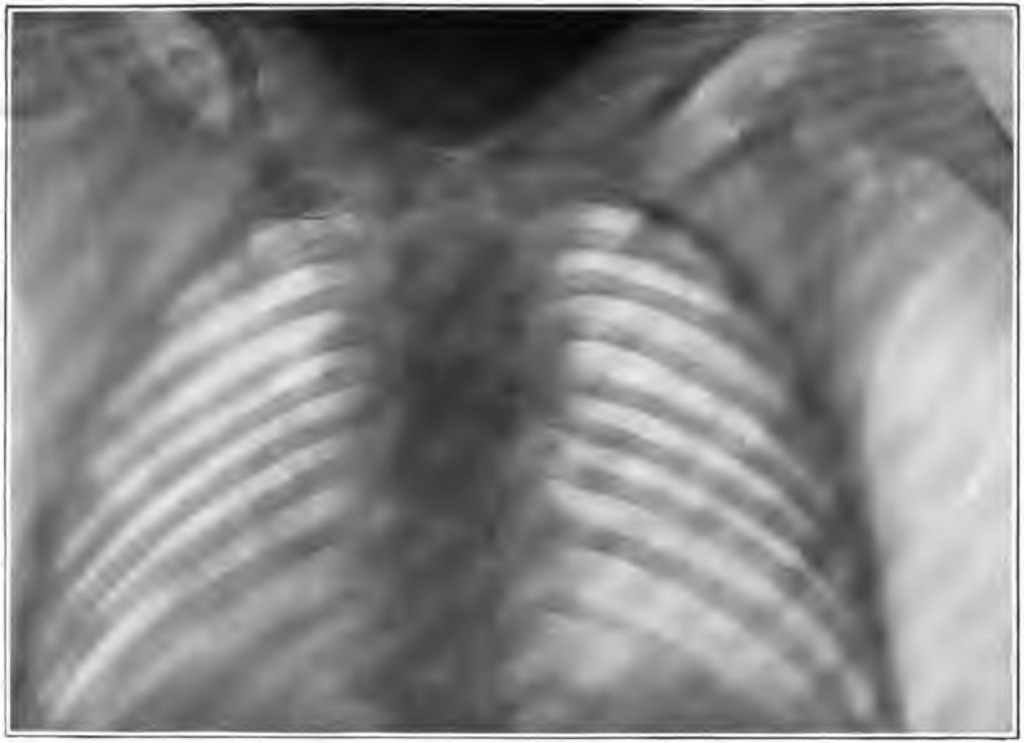

Fig. 3. *Skiagraph of the thorax of a child, aged 4 years, showing a disc-shaped dark area produced by tubercular infiltration.*

a very low vitality; the temperature was always subnormal. The patient had been kept recumbent during this period. To secure complete immobilisation the splint shown in Fig. 2 was applied. After she had worn the splint for about a month a slight improvement in voluntary movement and in the tone of the bladder was observed. This improvement was but temporary, and a few weeks later the muscular power in the left leg had become weaker, whilst the patient at night complained of pain in the legs. The motor paralysis gradually became absolute, the patient becoming rather weaker, and I felt that it was useless to wait longer for a spontaneous cure. The skiagraph,[1] reproduced in Fig. 3, helped me in forming the opinion that a prevertebral abscess was present.

Operation.—On January 15, 1902, eight months after the patient was admitted to hospital, I performed costo-transversectomy, removing the transverse process of the fifth dorsal vertebra and the neck of the coresponding rib.[2] I passed a director to the front of the vertebra and about a drachm of liquid pus escaped. I then passed a small Volckmann's spoon, and with it I carefully and gently stroked the fronts of the fourth, fifth, and sixth vetebral bodies,[3] and then the front aspect of the abscess-cavity where lay the œsophagus, &c. In this way I removed two drachms of tubercular matter. I then injected about the same quantity of a 10-per-cent. emulsion of iodoform in a mixture of equal parts of glycerine and 1–20 carbolic lotion, and inserted a small drainage-tube. The wound was then dressed and the child put back into the splint.

The day after the operation the drainage-tube was removed. The temperature was 99·8° F., and both lower limbs showed a slight degree of spasticity with exaggerated deep reflexes and ankle-clonus—a striking contrast to their previous flaccidity. This state of things continued for three days, and then everything returned to the condition present before operation. It appeared as if the operation had failed, so I resolved to reöpen and scrape the prevertebral abscess. This I did a week after the first operation. Nothing but a little clear fluid escaped, and no caseous matter came away in the spoon. I replaced the drainage-tube and decided to establish an aseptic sinus by keeping it in, for the double purpose of preventing any further collection of fluid and enabling the iodoform-glycerine-carbolic emulsion to be injected daily. The immediate result of the second operation was the same as the first—a spastic condition with exaggerated reflexes set in; this time, however, it did not so rapidly subside, and voluntary power began to be regained, the patient a week after the second operation being able, on request, to flex the limbs, but she could not extend them again; she appeared to be really frightened to move her legs. After this time the movement became gradually stronger, the left leg improving more rapidly. The sinus was allowed to close a fortnight after the second operation. The improvement was as steady as it was slow, and it was accompanied by a marked change in the patient's general health, the child becoming bright and less anæmic, and putting on weight. The power of extension gradually appeared in the limbs.

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[1] This was taken for me by Messrs. Coxeter and Son.

[2] In removing the portion of rib a minute puncture was made in the pleura: this I immediately covered with a pad of cyanide gauze, which was removed after four days.

[3] In the light of later experience (*see* Case D. and conclusions) I think this proceeding was too dangerous to be repeated.

On April 4, 1902, four months after operation, the patient could move both legs voluntarily, both in extension and flexion, the left still being the stronger: the exaggeration of the knee-jerk, &c., was now much less marked, as was also Babinski's sign.

For two months the limbs were treated by resisted exercises in bed, the patient being kept supine and wearing the splint. After this the thigh-pieces were removed, and for a month she was allowed to sit up occasionally in the daytime. In July she was placed on her feet and at first had difficulty in keeping upright even when supported. After a few days she could stand, and she soon began to walk, at first with the legs wide apart. By the end of August she could walk well and naturally, and appeared to be a normal healthy child; and up to the present no sign of relapse has been observed; about this time she was photographed sitting up without the splint (Fig. 4).

As the direct outcome of the foregoing case another reported by Dr. W. H. B. Brook to the Clinical Society of London (March 13, 1903) may be recalled. Dr. Brook was led to perform costo-transversectomy by seeing my patient at a previous meeting of the same society.

PLATE XV.

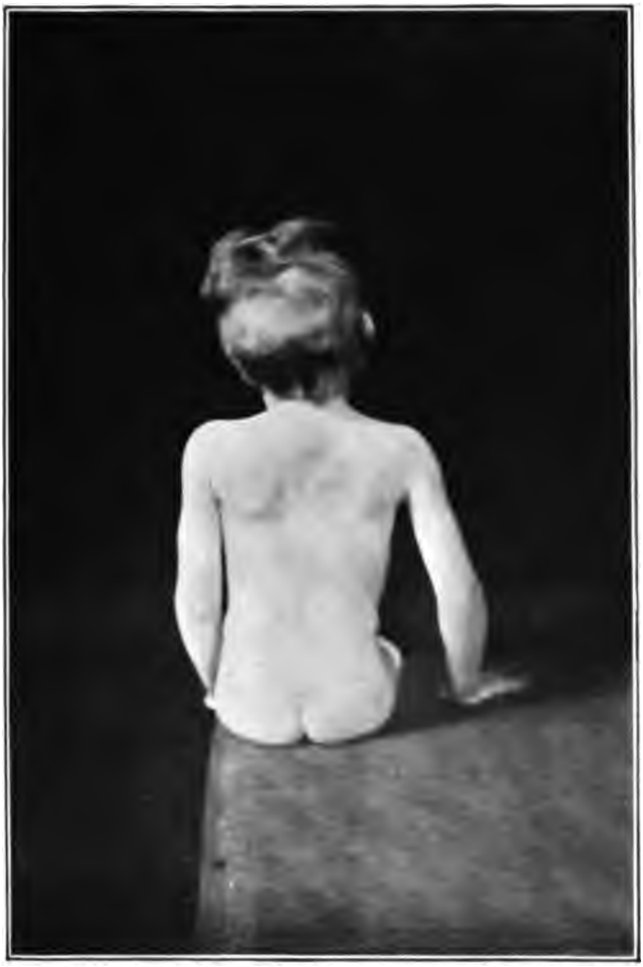

Fig. 4. *The back of a child, from a photograph taken seven months after a lateral operation for prevertebral abscess and paraplegia.*

Dr. W. H. B. Brook's case:—

Arthur A., a clerk, æt. 17, was admitted into the Lincoln County Hospital on September 22, 1902, suffering from angular curvature of the spine of one year's duration, for which he had worn a plaster jacket. During the previous four weeks he had gradually-increasing loss of power in his legs, with incontinence of urine. When admitted he presented well-marked angular curvature, the sixth and seventh spinous processes being the most prominent. There was no tenderness, nor where there any signs of any collection of pus. There was well-marked girdle-sensation at the level of the epigastrium, with great impairment of sensation below this level. The legs could be moved, but not well; the knee-jerks were exaggerated, and there was incontinence of urine. For six weeks he was treated by extension applied to each leg, but his condition became steadily worse, the paraplegia becoming complete, with incontinence of urine and fæces, and the appearance of bed-sores in spite of the greatest care. A skiagraph showed blurring in the region of the seventh and eighth dorsal vertebræ, confirming the diagnosis that tubercular matter was present. On November 8, the operation of costo-transversectomy was performed, the right transverse process of the eighth dorsal vertebra being cut down upon and removed, together with the head and neck of the eighth rib, so that access was obtained to the seat of the disease, and the broken-down bodies of the vertebræ were carefully scraped away with a specially-designed curved sharp spoon, care being taken not to wound the dura mater or the pleura. The wound was drained, injected with iodoform emulsion containing 1 : 40 carbolic acid, and lightly packed with iodoform-gauze. The extension was continued. Sensation began to return on the following day, and movement on the day after. At the end of three weeks sensation was perfect and movement good, but it was seven weeks before the patient regained perfect control over the bladder and rectum. The drainage-tube was discontinued on January 8; the wound was healed by January 25, and on February 15 the patient was able to sit up, after having been fitted with a steel spinal support. He is now able to walk quite well.[1]

The special spoon used by Dr. Brook is that which I had designed (Fig. 5). As will be explained later, the spoon, if employed at all, must be used with the greatest care. The steel support which Dr. Brook mentioned was Chance's splint as used at the City Orthopædic Hospital.

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[1] This abstract is taken from the *Med. Press and Circ.*, Mar. 18, 1903.

The case of another patient who came under my care at the City Orthopædic Hospital, and subsequently under the joint care of my colleague, Mr. Mayo Collier, and myself, at the North-West London Hospital, is worthy of record, since it illustrates some important points. I had previously fitted a Chance's splint to the spine.

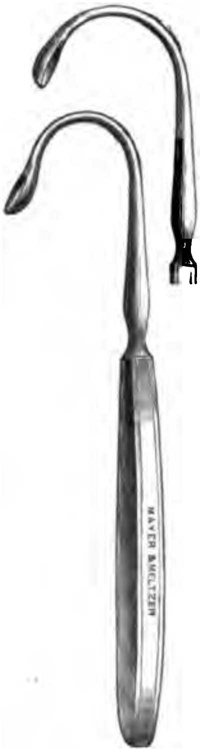

Fig. 5. *A Volckmann's spoon, the shank of which can be bent to any shape that is desirable.*

Case C.—A girl, aged 4½ years, of very weakly appearance. The spinal disease began at the age of six months, and paralysis rapidly supervened, so that the patient had been totally paralysed for four years. There was a rather sharp angular deformity in the upper dorsal region. The abdomen was greatly distended and tympanitic from paralysis of the abdominal muscles. The bowels were only relieved by means of enemata, and the urine flowed away from the paralysed bladder. The legs were cold and thin, and completely paralysed as to motor power. The knee-jerks were exaggerated; Babinski's sign was present. Ankle-clonus was occasionally present, with marked spasticity of the legs.

On November 19, 1902, Mr. Collier removed the right transverse process of the most prominent vertebra, and immediately about half a pint of pus escaped. A large abscess-cavity in front of the vertebræ was syringed out and lightly scraped; iodoform-emulsion was injected, and a draining-tube inserted.

On December 5, although the cavity was draining well, there was no improvement in the power of the legs. With Mr. Collier's consent, I removed the transverse process of the left side of the apex-vertebra. About two ounces of pus escaped, and syringing from one aperture to another across the abscess-cavity was readily effected. On December 11 the spastic condition of the legs was more marked than it had been before, and some doubtful voluntary movement was noticed. The condition of the abdomen began to improve, and the bowels acted on two occasions without recourse to enemata.

On January 14 the patient developed whooping-cough, and was sent home, the mother bringing her periodically to see me. A progressive, if slow, improvement was observed in the muscular power. The drainage-tubes were removed soon after the patient left the hospital, and the drainage-apertures healed. On August 13, 1903, in answer to my enquiry, the mother writes:—"My little girl's general health has never been so good (except that her bowels do not act naturally). . . As regards her legs, she has strong movement in them, and struggles to get on her feet, but directly gives way at the knees."[1]

A fourth case, in which death occurred from general tuberculosis shortly after the operation, should also be mentioned.

Case D.—A youth, aged 16. Eight months before admission to the North-West London Hospital he had noticed a projection at the upper part of the back. I had previously seen him at the City Orthopædic Hospital, and had applied a Chance's splint with a head-piece, from which he derived great comfort. Three weeks before admission the patient's legs became weak, and he could not walk without support, although he was able to raise his legs from the bed. There was spasticity in the legs. The knee-jerks were increased. Babinski's sign and ankle-clonus were present, Sensation was not impaired. The angular projection in the spine involved the second, third, and fourth dorsal spines. On January 5, 1903, I removed the third right transverse process, and at once came upon firm caseous material. A curved probe was carefully passed round the front of the spine, and then with a curved Volckmann's spoon more caseous material was removed, but no liquid pus was found. There was no shock or pain after the operation, but the degree of paralysis was increased. This increase of paralysis began to pass off, but symptoms of meningitis appeared on January 28, and increased up to February 8, when patient died quietly. An autopsy, carefully performed by

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[1] This patient is of course wearing an efficient support, and I must caution the mother against allowing these attempts at walking for the present.

Mr. R. J. Waugh, senior house-surgeon, revealed a large caseous focus at the seat of disease. The bodies of two vertebræ were completely destroyed, exposing the spinal cord, the dura mater also having been destroyed and replaced by caseous material.

Looking back on this case, I can only admit that the operation was performed too early, *i.e.*, before the tubercular material had undergone liquefaction. Tubercular meningitis is in my experience the commonest cause of death in cases of tuberculosis situated in the upper dorsal and cervical regions of the spine. In a case of this kind, where the dura mater had been penetrated by the disease, it was almost certain to occur sooner or later; but the operative interference probably accelerated its onset.

Reviewing the experience embodied in the above cases the following conclusions are arrived at:—

1. When paraplegia develops rapidly and involves sensory as well as motor functions, operative interference is necessary. Dr. Brook's case and my own first case are examples.

2. In other cases a patient trial of rest in bed with some form of extension should be tried. If this fails, a good skiagraph should be obtained. The choice is then between lateral drainage and laminectomy. The removal of a transverse process alone, when guided by the skiagraph, will be sufficient to open a large abscess (witness Mayo Collier's and my Case C.). It is safer to avoid any attempt to scrape out the abscess-cavity, for the cord may lie naked at the back of it (Case D.). When there is a sharp angular deformity in the spine, the cord will not be in danger of being bruised during the introduction of a tube, &c.; but when the spine is nearly straight the path for a drainage-tube should be made in a *forward* direction, for which purpose the Volckmann's spoon may be carefully used. Daily injections of a small quantity of carbolic lotion may be made daily through the tube. The transverse processes on both sides of the spine may be removed in order to obtain thorough drainage.[1]

Keetley's operation I consider to be too dangerous, in ordinary hands at least. Ménard's costo-transversectomy involves the risk of injury to the pleura (Case B.). If the removal of a transverse process does not reveal an abscess laminectomy

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[1] In cases of cervical disease with paraplegia, lateral drainage would be easily effected through an incision at the posterior border of the sternomastoid, as for retro-pharyngeal abscess.

PLATE XVI.

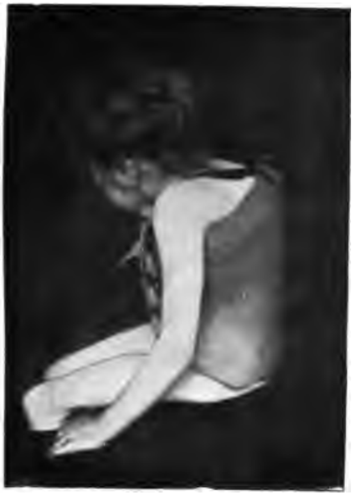

Fig. 6. *A child with high dorsal caries wearing a wrongly-designed "poroplastic" jacket.*

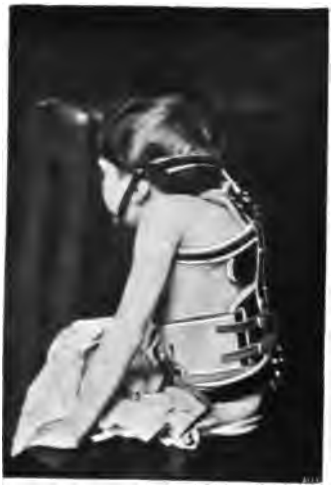

Fig. 7. *The same patient with Chance's splint applied.*

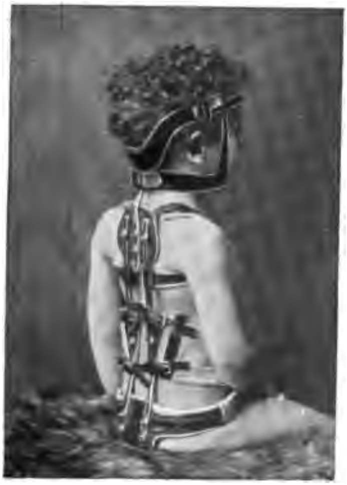

Fig. 8. *A boy with caries of the upper dorsal vertebræ wearing Chance's splint.*

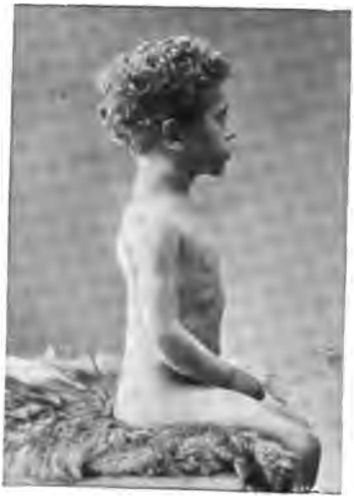

Fig. 9. *The same boy after two years' treatment.*

(For permission to use these four blocks we have to thank the Proprietors of the *Medical Press and Circular.*)

may be proceeded with, either immediately or after an interval; the presence of the lateral opening will provide an easy outlet for future suppuration, and probably prevent the unfortunate extension of tubercular infiltration around the spine (*see* above Case A., Lane's Case II.).

A few words may be added as to the mode of extension to be employed during the earlier part of the treatment. In every case where paraplegia is present in any degree, it is hardly necessary to say that the patient must be put to bed or on a prone couch. If bed-sores threaten, a water-bed must be used. Extension, wherever this is possible, should be employed in some form. Weight-extension has the drawback of confining the patients to bed and making it difficult to take them into the open air. Bradford's frame of iron gas-piping on which canvas is stretched, and on which the patient is easily carried, is better. Children bear the antero-posterior splint shown in Figs. 2, 7, and 8 very well; and the degree of extension thereby obtained is, in my opinion, more reliable than any other. This form of extension and fixation of the spine has the advantage of being ready for use without alteration, when recovery is sufficiently advanced for the patient to walk about; it is also adaptable to the growth of the patient, and so is less expensive than many forms of apparatus. As to the after-treatment, the "poroplastic" supports very generally used in this country must be noticed. There are very often applied only to reach up to the seat of the deformity, as shown in Fig. 6. Thus applied they give but little support; they hamper the respiratory movements, and so tend to impair the health and resisting powers of the patient; however frequently they are renewed they hamper the growth of the patient; they are hot, heavy, and impervious to perspiration. When antero-posterior splints, whether of the Chance or Taylor type, are used in cases of high dorsal or cervical disease, they should be provided with a head-piece—for instance, that shown in Fig. 8. With such an appliance the amount of deformity resulting from tubercular disease may be very slight or even *nil* (Figs. 4 and 9). Plaster-of-Paris corsets are even more harmful to the general health and development of children than those of felt. For temporary use after operation, moulded troughs of plaster of Paris or of felt for the patient to lie in, as recommended by Sir Victor Horsley, are useful.

RECENT WORK IN THE FIELD OF ANÆSTHETICS.

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The many contributions to the subject of anæsthetics that have appeared within the past year, although largely concerned with old fundamental questions, such as the physiological effects of chloroform and the manner of death under anæsthetics, yet touch upon new ground in one or two directions. Much, for example, has been written upon the comparatively new anæsthetic drugs of the ethyl-chloride class, of which more anon, and much attention has been paid to details of technique, not only in the actual administration of anæsthetics, but especially in the matter of proper preparation and proper after-treatment of patients subjected to anæsthesia. We do not propose in this review to do much in the way of criticism, but rather to put forward in abstracts or in actual quotations the various recommendations and theories that have been advanced by recent writers in this country, on the Continent, and in America. It is permissible, however, to remark that, judging at least from the literature of the subject, Great Britain is very well to the fore in the matter of anæsthetics. The scientific side of the subject, as might be expected, has been, and is being, as thoroughly investigated abroad, particularly in Germany, as it is here; but clinically it is obvious that methods discarded after long trial in England are still in vogue in foreign countries, whilst the latest advances in the practical administration of anæsthetics which have done so much in this country to dissipate patients' fears and diminish their discomforts, have not yet reached the Continent or America. So much is candidly admitted indeed by Dr. John Clark, Professor of Gynæcology in the University of Pennsylvania (*Therapeutic Gazette*, May, 1903), who declares that while "American surgery, so far as technical skill is concerned, is superior to that of England, they are much in advance of us in the administration of anæsthetics."

As regards the previous preparation of patients for anæsthesia and their subsequent after-treatment, Professor Oscar

Witzel, in the *Münchener Medicin. Wochenschrift* (December, 1902), describes a procedure more elaborate than any we have yet seen used or recommended. According to the Professor's view the undesirable lung-conditions sometimes following ether, the "ether-pneumonia" of some authors, are entirely attributable to infection acquired by inspiration, and are capable of being entirely avoided, if due precautions before, after, and during the administration are devoted to the antisepticising of the air-passages and to keeping them free from infection during the operation. For the latter purpose, Professor Witzel recommends in all operations a posture in which the neck of the patient is in a condition of over-extension, as in this way he says nothing can travel from the mouth to the air-passages, and mucus and saliva are continuously enabled to escape. "Lung troubles following ether-administration," —we quote Professor Witzel—"are due to liquid infected and flowing into the air-passages or sucked in from the mouth. They get less frequent with improved technique. We have had no death from lung-inflammation. Still there is often an acute catarrh of the larger air-passages. The cough-creating irritation of this catarrh is specially unfavourable after abdominal operations. None of this air-passage irritation need arise from ether *per se*, if we guard against it by careful technique and prophylaxis with regard to the air-passages, and if this is carried out also after operation. The steps to be taken are :—

i. Disinfection of mouth and air-passages before narcosis. The teeth are carefully cleansed, and the stomach is washed out before abdominal operations.

ii. Care regarding position during narcosis; the patient's head to be low and the neck in a position of over-extension.

iii. Ventilation of the air-passages by systematic respiratory movements after operation.

These are practised for days before the operation. Inhalations of salt-water steam and of turpentine during the same period complete the prophylactic steps—the most important features in the avoidance of lung-trouble from ether. Infarcts o. the lung caused by emboli from branches of the portal vein that have been tied will remain free from infection, if these steps are taken, and will be cured by simple "respiratory measures." The position of the patient here recommended is that which also afterwards. Vomiting was absent in all except one case, where the patient had taken solid food before operation. Lewin believes that vomiting after chloroform-anæsthesia is caused by direct irritation of the stomach by the chloroform-vapour which is swallowed. It then depends on the degree of irritability of the gastric nerves whether vomiting takes place or not. The vomiting which takes place after anæsthesia and often continues for several hours is caused by chloroform which has passed into the circulation, and is eliminated through the gastric glands. He suggests that the susceptibility of the gastric nerves may be previously reduced by means of a local anæsthetic, and that the gastric mucous membrane may be covered with a substance which will, to some extent, protect it from chloroform. He recommends the administration of cocaine in a small dose (1 decigramme) dissolved in sufficient water to flow over the whole of the mucous membrane. For the mechanical protection of the stomach against the action of chloroform, he recommends mucilaginous substances, which will adhere for some time to the walls of the viscus. The most suitable substances are one part of gum arabic to two parts of water and tragacanth. The patient should take a dose of this four hours before, and another dose immediately before, operation. In weak patients Dr. McCardie recommends preliminary enemata, once daily, of saline solution, subcutaneous injections of glucose (Barker), or nutrient enemata of egg, beef-tea, brandy and pepsin. As regards the air-passages, Dr. McCardie quotes the suggestion that in the case of patients suffering from cough advantage should be taken of the anæsthetic effect of oil of peppermint or menthol in the mucous membrane of the air-passages. Before etherisation Briggs (*Therapeutic Gazette*, June 1902) administered menthol to a nervous bronchitic patient with "magical effect." One drachm of oil of menthol is poured on to the inhaler, which is rapidly applied, and after six free inhalations the ether-administration is begun. It is claimed that there is no coughing, and that excitement is greatly lessened or prevented.

The question of the preliminary use of adjuvant drugs, particularly atropine and morphine, is engaging the attention of many anæsthetists at present. We have seen that in Germany it is in some schools the routine practice to precede ether by injections of morphine, when, as is there the case, the simple open method of giving ether is adopted. No doubt the assistance of morphine, at any rate in the case of difficult patients, must be of great convenience, if not absolutely necessary to avoid long struggling. With the closed methods in vogue in London the same cannot be maintained. As regards atropine, it may be observed that experimentally it has been proved to be of great value in preventing cardiac inhibition. Thus Crile, quoted in *Treatment*, March, 1903, says, "Atropine, hypodermically administered, is a sufficient protection against cardiac inhibition in operations on the 'inhibition area,' in the larynx, and in such operations as might cause mechanical stimulation of the vagi." The blood showed an unusual tendency to clot in his atropine-experiments. Cocaine, given hypodermically, guards the heart against inhibition almost as efficiently as atropine does. The reflex impulses in the laryngeal area of inhibition are wholly prevented by a ½-per-cent. solution locally applied, the action being pronounced in thirty seconds. Like atropine, cocaine seems to prevent splanchnic shock. As regards dosage of atropine in men, Binz gives the maximum dose as $\frac{1}{66}$ grain three times a day. Probably 1 or 1½ minims of the liquor atropinæ would be a sufficient dose for an adult in an emergency. Atropine suppresses laryngeal-reflex or primary syncope, and also secondary syncope. Respiratory syncope is not in itself fatal, and can be remedied by artificial respiration. Cardiac syncope, on the other hand, may rapidly be fatal. The use of atropine alone with chloroform (McCardie in *Treatment*, March, 1903) is not recommended, for (1) it does not allow of economy of chloroform; (2) it often causes phenomena of excitation. In man, Arthur recommends that "twenty to thirty minutes before inhalation of chloroform 1·5 cc. of a solution containing 10 cgr. of morphine and 5 mgr. of sulphate of atropine be injected." Morphine has been charged with producing respiratory syncope and collapse, and with increasing the nauseating effects of chloroform. The addition of atropine suppresses this difficulty. It has been said that $\frac{1}{7600}$ of a milligramme of atropine may cause noisy excitement, but morphine obviates this effect. It has been proposed to reduce the dose of atropine; but it is doubtful whether the smaller dose would paralyse the cardiac motor apparatus, or so weaken it as to obviate all danger of syncope.

The method of preliminary injection has also been blamed for not allowing full and quick awakening after operation. This objection can be applied to every method of mixed anæsthesia. Is it really a drawback ? Is not post-operative sleep reparative and rather an advantage ? Hewitt certainly has recorded peculiar and dangerous symptoms occurring after operation, and apparently due to the effect of morphine given while the patient was under the influence of the anæsthetic. He advises that the susceptibility of the patient to any drug which is to be injected before anæsthesia should be previously ascertained.

Many continental anæsthetists regularly inject atropine with morphine before beginning administrations of chloroform. Atropine, as is well known, paralyses the ends of the vagi in the heart, and besides removing the drag applied by these nerves, it directly stimulates the heart and in moderate doses keeps up the force of its systole. It stimulates the vasomotor centre and raises the blood-pressure. Thus stimulation of the vagus by chloroform or other means can be entirely prevented by atropine, and one important factor in the "early" chloroform-death should be capable of control (Emley).

McCardie (*loc. cit.*) adds ½ minim of liq. atropinæ to 15 of liq. strychninæ for hypodermic injection. In some cases he recommends in addition an ounce of brandy to be drunk just before administration ; this is a plan suggested by Mitchell Banks. Luchsinger, quoted in *Treatment*, March 1903. says: "If the heart's action has been brought to a standstill from the effect of chloroform. we can always at the first stage of the paralysis cause the heart to beat again, and that often powerfully, by administering atropine."

Another drug, the preliminary use of which is recommended, is adrenalin chloride. Martin of Pennsylvania (*Therapeutic Gazette*, May 1903) regularly uses a spray of "2-per-cent. cocaine in 1 to 10,000 adrenalin chloride." With this he sprays the throat and nose before giving ether, and believes that "the danger of cardiac and respiratory inhibition is greatly lessened and the secretion of mucus is much diminished." This writer declares strychnine to be actually harmful. He uses adrenalin chloride in normal salt-solution (1-1000) to combat shock. His belief in adrenalin chloride is based upon experiments of Reichert and Crile upon the dog.

The preparation of patients for anæsthesia is discussed in the same journal by Dr. John Kirk, Professor of Gynæcology in the University of Pennsylvania. He regards the "common cold either in its acute or waning stage as a very serious contra-indication to anæsthesia." He refers particularly to abdominal operations, when the risk of much coughing, such as might accompany an aggravated cold, would of course be highly undesirable. As regards preliminary examination, he lays great stress upon the condition of the urine, and considers that renal insufficiency with a small trace of albumen is much more portentous of danger than a considerable amount of albumen with an approximately normal quantity of urine. He looks upon albumen and casts not as a serious contra-indication for anæsthesia unless there is marked deficiency in the watery constituents. He is convinced that in skilled hands ether rarely produces a serious kidney-lesion. He believes in the ingestion of increased amounts of fluid up to two hours before operation.

Turning from questions primarily concerning the patient, such as preparations and after-treatment, to the question of the anæsthetic drug itself, we find a considerable quantity of recent literature. The undying argument as to the preferability of ether or of chloroform is as freely sustained as ever. We do not propose to quote from the combatants at all; the merits, demerits, and proper use of these two cardinal anæsthetics have been by now made fully familiar to all medical readers in this country. For the same reason we shall not deal with new methods of regulating dosage of chloroform such as that described by Professor Vernon Harcourt. Generally speaking, continental views uphold our own, though the methods of giving ether are often dissimilar. The modes in common use in England are, however, well known and described in recent text-books (e.g., *Vari Metodi Anestesici e loro Indicazioni*, by Giovanni Palleroni, of Palermo).

The most important contributions to this branch of the subject, viz., what anesthetic to use, centre round the comparatively-new drug ethyl chloride and similar substances.

The principle upon which were based the hopes of great success from these bodies is that most prominently advanced by Schleich, and deals with the relation between boiling-points of anæsthetic drugs and the temperature of the human lungs. The more volatile an anæsthetic, the less is absorbed by the blood in a given time and the more rapid is its elimination. The more closely the boiling-point of the anæsthetic drug approaches the temperature of the blood in the capillaries of the lungs, the more completely will inhalation and elimination, with the aid of expiration, balance one another, so that no accumulation of the anæsthetic takes place in the body. Schleich believed that by his mixtures he had attained the object of finding a respirable anæsthetic, whose effect was perfect, and whose elimination was as near as possible immediate. Dr. W. Meyer (*Journal of the American Medical Association*, February, 1903) has worked along the lines of Schleich, and especially with a mixture that he calls "anesthol." This mixture consists of 83 per cent. of a molecular solution of chloroform and ether and 17 per cent. of ethyl chloride. As regards this "molecular solution," Dr. Meyer states that, as a result of experimental analyses of Schleich's mixtures, which were of chloroform, ether, and petroleum ether, Dr. H. P. Weidig and himself (Meyer) have come to the conclusion that chloroform and ether, when mixed in the proportions of 43·25 parts of chloroform with 56·75 parts of ether, enter into chemical combinations forming a new anæsthetic, a body containing neither free chloroform nor free ether. It will be observed that these proportions are almost exactly those commonly used in some London schools, where the A.C.E. mixture is often employed without its alcohol. This C.E. mixture is recommended both by Hewitt (*Anæsthetics and their Administration*) and by the present writer (*Anæsthetics*, Medical Monograph Series), who have used it in large numbers of cases. Dr. Meyer points out that he is not claiming to have discovered in this combination of ether and chloroform a new body in the sense in which, for instance, acetone and chloroform combine to form one. He maintains, however, that when ether and chloroform are mixed in the above-mentioned proportions the combination has an evaporating-point of its own, and will resist fractional distillation to the last.

Assuming the correctness of Schleich's principles, Dr. Meyer sought so to modify this "molecular solution" that its boiling-point from 125·6° F. should be reduced to a point the same as, or near to, that which represents the temperature of the blood in the pulmonary circulation. Anesthol was finally arrived at, with a boiling-point of 104° F. It is described as a clear, transparent liquid, of agreeable odour, with specific gravity 1·045. It is a chemical combination of the above-mentioned "molecular solution" and ethyl chloride, not a mixture. Thus 1,000 cc. of anesthol were evaporated for five hours at a uniform temperature of 140° F.; 22 cc. of fluid with sp. gr. 1·262 were left. The sp. gr. of chloroform is 1·490. This fact proves that, up to the last moment, a "solution of components" discharges, and does not leave a final residuum of chloroform, the narcotic with the highest boiling-point, as is the case with Schleich's mixture.

Ethyl chloride as a general anæsthetic is particularly recommended for short operations in children (Dr. W. J. McCardie, *Lancet*, April, 1903). The induction of anæsthesia is very rapid. In most cases excitement varies directly as the quantity of air admitted during the induction of anæsthesia. It is said that in Russia, where people are great consumers of exciting drinks, especially alcohol, tea and coffee, excitement is much more common than elsewhere. Blank of Kieff accuses immoderate tea-drinking of predisposing to excitement, and excessive use of coffee to vomiting. Dr. McCardie uses 60-cc. graduated tubes of ethyl chloride from which he sprays the drug on to the sponge of an Ormsby's inhaler. Three to five c.c. are the dose generally used from the beginning. The average duration of induction was 50·9 seconds, average duration of anæsthesia 71·3 seconds. The shortest anæsthesia met with was 3 seconds, and the longest 2½ minutes. The opinion of those who have operated upon cases of adenoids and tonsils under ethyl chloride (in Birmingham) is favourable. There is rarely difficulty in obtaining complete muscular relaxation. Headache and sickness sometimes follow, much more commonly than is the case with nitrous oxide. McCardie records four severe cases of vomiting in 450 cases.

Along with ethyl chloride may be mentioned the much-advertised anæsthetic "somnoform," the effects of which depend mainly upon the 65 per cent. ethyl chloride which is its chief constituent. Experience with this anæsthetic at the hands of various anæsthetists was put before the Society of Anæsthetists (*Lancet*, April 25, 1903), where Mr. W. Foster Cross read a paper on the subject. There appeared to be a very general agreement that there was no advantage in somnoform over pure ethyl chloride, the results of their use being very similar; and the latter is preferable as a pure and simple drug without a patent name. In a preceding number of *The Lancet*, Mr. Eastham gives a brief account of bichloride of methyl ethylene, which under the name "narcotile" seems to play a part very comparable with that of somnoform. He employed a Lobjois inhaler, whereas that so often and so inefficiently used with somnoform is Brewer's.

In the *Medical Record*, May, 1903, Dr. H. H. Edrett records a case of "total respiratory failure from ether," with recovery after four hours' artificial respiration. The case is that of a man, aged 20, operated on for very advanced acute appendicitis with general peritonitis. Respiratory embarrassment was first noticed after flushing out the abdomen with salt-solution. Strychnine ($\frac{1}{80}$ gr.) was injected hypodermically and 30 minims of adrenalin chloride were given by the mouth. Respirations became progressively slower, until ten minutes after the first-mentioned symptoms were noticed breathing stopped. Artificial respiration was kept up continuously for 4 hours and 20 minutes. During this time it is stated that "at no time after the first dose of 30 minims of adrenalin chloride did the action of the heart cause any anxiety." Nevertheless, an extraordinary variety of treatment was tried, including dilatation of the sphincter ani. Adrenalin chloride was administered for a day after operation in doses of 10 drops every two hours. The patient finally recovered completely.

A CASE WITH COMMENTS.

A CASE OF HENOCH'S PURPURA.

By JOHN ORR, M.D., F.R.C.P.E.,
Physician to the Western Dispensary, Edinburgh.

CASES of purpura, of the type described by Henoch, are not of very frequent occurrence in clinical records, and it is with a view to draw attention to this interesting, and at first sight puzzling condition, that I venture to publish notes of a case.

The family, of which my patient was the fifth child, has had a very varied record of illness. Her father has on several occasions suffered from rheumatic fever, and more than once his fever has been remarkably obstinate and intractable. As one would naturally expect, there has remained considerable damage both to his mitral and aortic valves. During none of the attacks of rheumatic fever has there appeared a trace of any tendency to hæmorrhage, subcutaneous, cutaneous, or mucous. The patient's mother is stout, strong and healthy, and comes from a stock rather noted for the longevity of its members. The patient had originally four brothers and five sisters. Of these two brothers, the eldest and the youngest are dead of tubercular affection of epiphyses and joints (at elbow and at knee respectively), complicated by secondary waxy disease, and, in the case of the youngest brother, by multiple tubercular intracranial tumours. Another brother died, at the age of 15, of the effects of mitral valvular disease, apparently the result of a rather vaguely described attack of rheumatic fever in childhood. All the patient's sisters have enjoyed an almost total freedom from disease.

A. W., the patient, was 14 years of age. Without having any definite organic defect, she has been rather a delicate child. Her teeth are very soft and carious, and in consequence her food is not well masticated. A year prior to the illness to be presently described, she suffered from Purpura simplex rheumatica. The little petechial hæmorrhages came out in successive crops on both ankles and legs, and were variably preceded by considerable local pain. The pain was referred to those areas of skin where soon afterwards the petechial extravasations appeared. After about ten days the condition disappeared, and most benefit appeared to occur subsequent to the administration of sodium salicylate and 5-minim doses of turpentine. Slight arthritic, but no visceral or pyrexial, manifestations were present during the illness.

A year later, when the patient was 15 years old, a condition at first sight to all appearance similar to the above began, but speedily developed additional and more serious features. At the outset there was no constitutional disturbance; but malaise and a distinct feeling of illness supervened, and the temperature and pulse rose slightly, to 99·6° F. and 106 respectively. Moreover, in addition to the petechiæ about the wrists and ankle-joints, there appeared patches of an erythematous nature on the extensor aspects of the legs. At intervals of four or five days there occurred exacerbations of the constitutional symptoms, but no visceral complications of any sort appeared. The wrists and ankle-joints were slightly affected with pain and swelling, but not to any great extent. The patient was kept strictly to bed, and placed on milk-diet; but this regimen, in conjunction with the use of sodium salicylate, alkalies, and turpentine, had really little apparent influence on the disease. After a fortnight the clinical phenomena became absolutely periodic, and some new features developed themselves. The tongue assumed a most peculiar appearance, which I can only liken to what I have seen in cases of psilosis, although it was by no means exactly the same in all respects. Its surface became covered in figurate patches, oval in some places and irregularly-shaped in others, with a whitish fur, which occupied a reddened surface from which the superficial epithelium was detached. Unlike sprue, however, there was not the least undue sensitiveness of the epithelium-denuded areas. The sides and dorsum of the tongue were both involved in this manner. Simultaneously there occurred considerable abdominal pain and vomiting of bilious matter and of mucus, attended by nausea and depression of spirits, and for some time it was a matter of much difficulty to get any form of nourishment retained. Bearing-down pains and loose stools accompanied the sickness. At first the stools were merely abnormally fluid, and were of normal colour. The temperature now rose to 101° F., and oscillated between that and

102·4°, with a pulse of 110 to 120 per minute. In two or three days the gastro-intestinal derangement ceased under the influence of bismuth salicylate, and for a few days the patient became rapidly brighter, and improved in strength. Then there ensued a remarkably periodic recurrence of all the symptoms, the cycle of events lasting a week. Firstly the tongue, which had become practically normal in appearance, became covered by the patchy arrangement of fur as already described; next day the temperature and pulse rose; and these symptoms were followed by vomiting and loose motions. This chain of events recurred with remarkable regularity for six weeks. During two of these weekly exacerbations of the disease the patient passed a large quantity of fluid blood by the rectum, and rectal examination failed to detect any local cause for this hæmorrhage, such as ulcer, hæmorrhoids, or polypoid growths. The blood was reddish-brown or quite red in colour, differing a little on the two occasions, and was not clotted. There were no hæmorrhages from any other mucous surfaces. I regret that no examination of the blood was made. The urine was free from albumen, and never contained blood. No recurrence took place after the sixth week.

The treatment adopted was the usual dietetic treatment for rheumatic manifestations, and the diet consisted largely of milk diluted with potash-water, and with a little sodium bicarbonate and sodium chloride added to it, to favour the formation of a soft curd and to prevent undue constipation. Salicin, salol, and bismuth salicylate along with turpentine were also exhibited, but without any decided beneficial results. After remissions began to occur regularly, I tried the effects of giving abundantly the juice of oranges, and it is noteworthy that the use of this was followed by decided improvement, and finally by entire cessation of the recurrences. Whether the improvement was "*post hoc*" or "*propter hoc*" I am unwilling to say, but in future it would be part of my treatment to give orange-juice freely from the first.

These clinical features correspond with the peculiar variety of purpura described by Henoch in his *Lectures on Diseases of Children* (translated for the Sydenham Society by Dr. John Thomson). The disease appears to be associated with or to complicate certain cases of Purpura simplex rheumatica, and is characterised by the occurrence of moderate or slight, painful, articular swellings and gastro-intestinal disorder, such as pain and uneasiness in the epigastrium and lower abdomen, with vomiting and diarrhœa, and hæmorrhages from the bowel. It would appear, too, that relapses are common, and, in the case I now record, the relapses were strictly of seven-day periodicity. The exacerbations invariably occurred on the same day of the week (Tuesday), and gradually the patient improved, till by the Friday or Saturday at latest she was to all appearance almost well. Then on the Monday the tongue began to be characteristically furred, and next day she was as ill as ever, and the whole train of symptoms was gone through again. The periodicity was so remarkably exact that one ceased to be disappointed when, without any apparent cause, the patient, convalescing favourably, again became prostrated by a relapse.

Cases of rheumatic nature showing hæmorrhages are quite commonly met with, but it is not usual, I think, to meet with articular cases where hæmorrhage occurs from mucous surfaces, and where such peculiar features, as are above related, appear. What the cause of this malady is, it is very difficult to say. It may have some association with such diseases as hæmophilia and Werlhof's purpura, but this is not proved. It would almost seem that there must be some condition present in addition to the rheumatic taint, which is capable of producing extravasations from the blood-vessels, subcutaneous and mucous. That the cause of this hæmorrhagic tendency is not one associated with organic changes in the vessel-walls, such as endarteritis, seems a matter of comparative certainty, because the disease is an acute febrile one, and after a course of more or less short duration, disappears completely; nor can one detect any organic change in the vessels which are accessible to palpation. It may be of toxic origin, and the alimentary canal—which is evidently much disturbed in Henoch's purpura—may be the site of formation of the toxine. On the other hand, it is possible that toxines produced by the diplococci of acute rheumatism may be the active causal agents, but on this point we have as yet no definite information.

The disease is one of much interest, and a ready recognition of it may lead to further clinical investigation, by which our knowledge of its nature and causes may be increased.

THE MEDICAL PROFESSION FROM A FRENCH POINT OF VIEW.

A RECENTLY-PUBLISHED volume by Professor Brouardel,[1] dealing with the present position of the medical profession in France, is of considerable interest, as affording a comparison of the conditions prevalent there with our own problems. We find that the grievances of which our French colleagues complain are curiously similar to those met with here.

In the first place we are met by a complaint that the number of the profession is sadly in excess of the means of gaining a livelihood, and that this number is increasing yearly. The reason given by Dr. Brouardel for this state of affairs is that public interest in all branches of medicine has enormously increased of late years, medical subjects now constantly finding a place in the lay papers, whereas such a thing was practically unknown a generation or two ago. Parents are thus led to send their sons into the profession, owing to their having it so much before their eyes. The great discoveries made in recent years have also tended to popularise medical subjects. Such reasons may have something to do with the increasing number of doctors; but we fancy there are other causes connected with the spread of education and the desire to avoid any but "genteel" forms of occupation, which have a distinct bearing on the subject, in this country at least. The wish to avoid military service is also assigned as a predisposing cause in France.

Not only is the number of doctors increasing, but that of patients is diminishing. This results from the attention now paid to matters of public health. Many preventable diseases are thus actually prevented, and in this way the total amount of disease in the community is much reduced. Statistics show that in Paris there were 100,000 less sick persons during the year 1900 than in 1886, while the number of doctors has meanwhile increased from less than 1,900 (there were 1,963 in the year 1893, and the numbers for each year are slightly in excess of those for the previous one) to 2,846 in the year 1900. The figures speak for themselves as to the increased difficulty of making a living.

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[1] *La Profession Médicale au Commencement du XXe. Siècle.* By Paul Brouardel, Professor of Legal Medicine at the Faculty of Medicine, Paris, &c. 1903. Paris : Baillière et Fils. Price 3 fr. 50 c.

Hospital abuse is not confined to this country ; it exists also in Paris. Of a total of 60,000 *accouchements*, 27,000 were attended either in institutions or by midwives employed by the "Assistance Publique." Certainly the proportion seems enormous. The remainder were divided between 3,700 practitioners, including doctors, midwives, and "Officiers de Santé."

We find our neighbours over the Channel discussing also the vexed question of a classical, as opposed to a scientific or utilitarian, basis of education. On this point we are pleased to find that Professor Brouardel has strong views, and that they are in favour of the older method of mental culture. A doctor must, as he points out, have scientific training to enable him to carry on the technical side of his work ; but this is not by any means the end of his duties. He must not only be able to diagnose a disease and to prescribe the necessary remedy, but he has also to control and impress his patients ; and to do this, he must show that he possesses superior or at least equal intellectual attainments. The doctor must then have the best possible education that is to be got. He must not be inferior to his patient in literary or general knowledge, or he will lose his prestige. To quote from Professor Brouardel, the doctor " must impose his will on the sick man and his *entourage*. He must have authority and ability to convince. One of the sources of his authority comes from the superiority of his intellectual culture." In this connection some quotations are given from an article by M. Alfred Fouillée ; among them the following : " Many moral and even social problems are presented to him (the doctor) in the practice of his profession ; he has often to face formidable responsibilities. He is for many families a counsellor in a thousand difficult situations. He frequents the most cultivated society at the same time that he devotes himself to the poorest patients ; he ought to have himself received the highest moral culture, if he is not to be reduced in the eyes of his patients to the level of a mere tradesman, a dealer in life and death (*un industriel, un exploiteur de la vie et de la mort*) . . . It is not a question of knowing if Greek and Latin are or are not necessary to an understanding of medical terms; what a miserable way of looking at things on their most paltry side! The question is whether the highest culture is not needful for the most exalted mission, and whether Society has not the right, in order to protect its own members against quacks and charlatans," to exact the best possible literary and philosophic education from all doctors.

The value of a literary education is supported by other witnesses. Thus the celebrated Liebig, a great upholder of a scientific training, wrote that "pupils coming from the *Realschule* to my laboratory are, during their first year, superior to those from the *Gymnasia;* in their second year they are equal; in their third year they fall behind them." (The *Realschule* afford a purely scientific education: the *Gymnasia* give a classical training.) With regard to those who took a degree of Doctor of Science, M. Lippmann says that: "After they have taken their degree of doctor, they work no more, they make no discoveries; under guidance they progress well; but they have not the mental training (*gymnastique*) to walk alone. Their initiative is at fault."

Professor Brouardel criticises the medical education of the United States, on the ground that the course is too short, and that the students enter on their work without the possession of sufficient preliminary culture properly to appreciate the technicalities of the art. "The result is, according to the statements of American professors, that they turn out good surgeons, but that they are inferior in internal pathology and medicine." Hence there is a movement in America in favour of a longer course for medical students, and of better preliminary preparation as a basis. Professor Brouardel is in favour of the inclusion of Latin and modern languages in this preparatory course, rather than Latin and Greek.

Professional ethics are now the subject of lectures at the medical schools in France. The subject is spoken of as "Deontology." There, too, as in England unfortunately, it is found that the ancient adage as to two of a trade not agreeing is applicable to the medical profession as well as to humbler pursuits. Professor Brouardel sighs over the fact that "the young men show some indifference toward their elders, and the elders look with displeasure on young men, full of new ideas, coming and setting up at their doors. Hence arises a strained situation, dissensions, and open or secret quarrels, in which the two doctors have everything to lose and nothing to gain."

But it is not only on the side of the doctors that "Tempora mutantur, nos et mutamur in illis." Patients, too, have altered for the worse. They no longer remain content with one medical attendant, as used to be the case. The trusted family physician is an institution of the past. Now the patients run about from one doctor to another, getting different prescriptions from each, and either taking none of them, or else any one which may most strike their fancy. The medical attendant no longer recommends the specialist to whom the patient is to go. The latter chooses one for himself, or more probably goes to a different one for each of a dozen ailments. Thus Professor Brouardel was asked to advise as to a specialist for the headaches of a child, and on questioning the parent found that the boy was already going to one specialist for indigestion, and another for a slight exostosis of the tibia. Another patient with a trivial affection of the scalp had been to get advice from four specialists before coming to Dr. Brouardel, which he appears to have done without trying any of the previous prescriptions.

On the whole it is comforting to recognise that it is the same in all countries, and that we are not alone in suffering the evils which strike us so forcibly in medical practice in this country. Social conditions are everywhere changing, and the medical profession must adapt itself to its environment. Numbers will no doubt ultimately right themselves, in accordance with the laws of supply and demand, but there may be a good deal of suffering to individuals in the process. The public will, perhaps, in time come to see that rules of professional etiquette are in their interest as much as in that of the doctor, and will learn wisdom in their relations with professional men. They may even—towards the end of the Millennium—realise that qualified medical men may be better to trust to than quacks and charlatans; but it is well not to be too sanguine.

W. C. B.

Reviews of Books.

Diseases of the Intestines. By JOHN C. HEMMETER, M.D., Ph.D. In two volumes. With many illustrations, some coloured. Vol. I., pp. 742. Vol. II., pp. 679. London: Rebman, Limited. Price of the two vols., 50s.

THE author's name is so well known as an authority on diseases of the stomach and the disorders of digestion that the reader is not surprised to find that the kindred subject of diseases of the intestine has been treated with the same exactness and completeness. The work is so large, and deals with the subject-matter with such minuteness, that it is more like a system than an ordinary treatise on intestinal diseases. Though the great bulk of the work has been written by Dr. Hemmeter, he has embodied contributions on four more technical or special subjects, viz., the Anatomy and Physiology of the Intestines, the Examination of the Urine and Fæces, Intestinal Bacteria, and Diseases of the Rectum. Of these the first three are from Associate Professors in the University of Maryland, and the last from a Professor of Proctology (!) in the Cleveland College.

The first volume opens with a brief description of the anatomy, the best feature of which is the coloured diagrams of the structure of the intestinal wall taken from F. P. Mall. After a fuller account of the physiology, the bacteria of the intestine are considered, and the reader will find a useful account of the colon-group and of the relationships of its members. This section contains a number of useful plates, among which are microscopic fields from positive and negative agglutination-tests. The careful and thorough examination of the fæces is insisted upon as being quite as essential in intestinal disease as is the examination of the sputum in respiratory affections; and practical directions for this neglected study are given. An account of the author's method of obtaining the contents of the small intestine by intubation of the duodenum is illustrated by figures of the apparatus and the way in which it is employed. A special section is devoted to the general treatment of intestinal affections; it includes a brief summary on subcutaneous feeding, and gives some details of a case in which the hypodermic injection of sterilised olive-oil was carried on daily for three weeks with benefit. The various intestinal diseases, beginning with diarrhœa, constipation, and colic, and going on to dysentery, ulceration, and new growths, are fully and systematically considered in Volume I. The lesions are well illustrated by well-executed figures.

In the second volume the author deals with subjects which are many of them surgical in essence and require operative treatment. The same clear special descriptions are to be found in this part as in the former; and surgeons who are much engaged in abdominal operations will find it a useful and valuable book of reference.

The first subject dealt with is appendicitis, and Hemmeter rightly insists on the immense difference, both pathological and clinical, that obtains between a simple appendicitis and the suppurative variety. Even in the former he has to admit four groups, which he terms Endo-appendicitis, Parietal-appendicitis, Peri-appendicitis, and Para-appendicitis, the last being usually suppurative. Whilst we may fully admit the desirability of picking out typical groups of cases for dogmatic descriptions, yet in no other disease is it so difficult to associate the clinical and pathological signs together, and hence too great an insistance on a mere pathological classification is unnecessary. Hemmeter makes no error in this direction, when he reaches the clinical side of his work, and all these pathological niceties are cast aside. The description of the symptomatology is on the whole good, but to a surgeon's mind is a little vague here and there. In particular, we note that, although he admits that a temporary improvement may follow an acute outbreak before further pyrexia ushers in the suppurative process, yet this is looked on as unusual. A surgeon, on the other hand, sees quite a number of such cases; and if he be called in at that stage, finds it often difficult, and usually impossible, to be certain as to what will happen in the next forty-eight hours. As a means of diagnosis, exploratory puncture has been advised by some German authorities, but Hemmeter shows correct surgical acumen in condemning the practice. At the same time his section on Diagnosis is defective in not containing any reference to a blood-count. The indications for operative treatment are on the whole accurately given, but the section might with advantage have been considerably elaborated. His general statement is as follows:—"If the disease symptoms, such as fever, acceleration of the pulse, pains, and the condition of the abdomen in general, do not improve in two days after their beginning, but continue with undiminished intensity, or if an aggravation of symptoms follows a considerable improvement which took place in this space of time, then an operation ought to be performed at once." Other indications are also given, but we cannot refrain from noting the absence of one most important and grave sign, viz., a rapid fall of temperature with a rising pulse, especially if associated with hiccough.

In the next section Hemmeter deals with tuberculosis of the intestine, and discusses briefly Koch's dictum as to the non-transmissibility to the human subject of bovine tuberculosis. His opinion certainly inclines towards an opposite conclusion. Three types of tubercular affection of the intestine are described, viz., tubercular enteritis or colitis, tubercular ulcer, and the tubercular neoplasm of the cæcum. The diagnosis between the last affection and cæcal carcinoma is carefully discussed, considerable stress being laid on the slow and late onset of stenosis in tubercle, as opposed to its early and rapid development in cancer.

Syphilis and actinomycosis are also dealt with, and then the *pièce de résistance* of the work is reached, viz., intestinal obstruction, to which nearly 300 pages are allotted. Nothnagel's phraseology is followed in this section, and until one has got clearly into one's mind the limitations accorded by him to such terms as stricture, constriction, stenosis, occlusion, obturation, &c., it is a little difficult to follow. The whole subject is dealt with in a masterly and methodical manner, both from the pathological and the clinical points of view, whilst special attention is devoted to diagnosis.

The surgical treatment of these various conditions is displaced from its natural surroundings and located in a chapter by itself—a plan which does not commend itself to us. In the first place he deals with appendicitis, and divides it from the clinical point of view into eight groups. His ideas are somewhat more conservative than will be found in most American books, and we have but little fault to find with his conclusions.

He rightly emphasises the difficulty of determining the existence of pus, but admits the ready curability of many cases by simple medical means. The collaboration of an experienced surgeon with a physician in all cases is recommended, and "let the patient be seen three to four times daily."

An unusual feature of the book is the closing chapter, which deals with rectal surgery, but in so summary a fashion that, *quâ* surgical treatment, it is of little value. Of real worth, however, are the sections devoted to physical examination and the diagrams illustrating the use of a speculum and the appearance of Houston's valves.

In conclusion, one is forced to express admiration for this very thorough work on Intestinal Diseases. The diction is clear, the arrangement (with one or two exceptions) good, and the material carefully prepared. It is needless to mention that it is well got up, and efficiently, though not excessively, illustrated.

A Text-book of Medical Jurisprudence, Toxicology, and Public Health. By JOHN GLAISTER, M.D., Professor of Forensic Medicine and Public Health in the University of Glasgow. Pp. 820. 238 illustrations. Edinburgh: E. and S. Livingstone. 15s. net.

THESE subjects naturally go together, and it is a great advantage to have a text-book of convenient size which gives a full and satisfactory account of both forensic medicine and of hygiene like Dr. Glaister's present work. The text, especially that portion dealing with drainage, is well illustrated by clear figures which explain the working of traps, ventilators, &c., and add to the value of the work. Though admirably adapted for students there is much that will be of great use to medical officers of health and those engaged in medico-legal practice, the more detailed portions, such as cases and methods, being put into smaller print. The volume is well got up and should certainly be a success.

The Study of the Pulse, Arterial, Venous, and Hepatic. By JAMES MACKENZIE, M.D. Fig. 335. Pp. 325. Edinburgh and London: Young J. Pentland.

THIS is an original study representing the results of an enquiry which has engaged the author's attention for the last 20 years, but—and this is not the least remarkable fact about the book—the work has been carried out in the intervals of general practice and not in the quiet of an endowed laboratory. The work is divided into three parts, the first deals with the arterial pulse and the movements of the heart, the second with the pulsation of the veins and liver, and the third with the venous and liver-pulses in irregular action of the heart. Dr. Mackenzie has done much of his work with an instrument, the clinical polygraph, which enables tracings of the apex-beat, and of the radial, carotid, venous, or hepatic pulse to be taken at the same time. It consists of a cup or "receiver" from which the pulsations are communicated by a tube to a tambour attached to a Dudgeon or Jacquet sphygmograph. This instrument is more delicate than the hand, as shown by the statement that it shows that all enlarged livers due to backward pressure of blood in heart-failure pulsate. The original observations are numerous, and the author does not bow the knee to tradition in his work, which marks a distinct advance in knowledge.

The Diagnosis of Surgical Diseases. By Dr. E. ALBERT, late Director and Professor of the First Surgical Clinic at the University of Vienna. Authorised Translation from the Eighth Enlarged and Revised Edition by ROBERT T. FRANK, A.M., M.D. (New York). Pp. 419. 53 illustrations. London: Henry Kimpton. Price 18s. net.

THIS volume presents to the reader a series of articles on the clinical diagnosis and recognition of a variety of conditions which are grouped together on a regional basis. There are but few works in which this is attempted from the surgical side, although many volumes dealing with medical diagnosis are available. Certainly this book can scarcely be placed on the same level as many of these. We are afraid that a somewhat ancient edition has been utilised for the purpose of translation, and hence many recent methods are not alluded to at all, *e.g.*, the Röntgen rays or the examination of the blood. Such as it is, there is a large amount of useful clinical material, and no one can thoughtfully read it without deriving benefit therefrom; but to call it systematic or thorough would be quite inaccurate. Thus in the first few chapters dealing with head-injuries and their complications, there is scarcely any reference to the cortical centres and no diagram or figure to illustrate their position, without which such articles are of little value; moreover, the term Optic neuritis or its equivalent does not appear in this section. It is obvious that such a patchy work as this demands but little attention, and indeed, we consider that the translator could have been better engaged than in its production. All the same, he has done his work well; the translation runs easily, and the ripe experience of a man like Professor Albert is not to be disdained, even if it be not up to the latest standard.

The Edinburgh Medical Journal, edited by G. A. GIBSON, M.D., F.R.C.P. Edin., and ALEXIS THOMSON, M.D., F.R.C.S. Edin. New Series: Vol. XII., 1902, and Vol. XIII., 1903. Edinburgh and London: Young J. Pentland.

THESE volumes contain much excellent matter, maintaining the high level usually reached by our Scottish contemporary. Of the original articles, the Morison Lectures on "The Nervous Affections of the Heart" by Dr. Gibson are of special value. In Vol. XII. Dr. Coutts' paper "On the Occurrence and Treatment of Lobar Pneumonia in Young Children," and Dr. Latham's on "Some Points in the Ætiology of Tuberculosis," deserve the permanence given them in this Journal. Of the various papers dealing with newer material, Dr. Bonney's careful investigation of 100 consecutive patients with reference to "Injurious Renal Mobility in Relation to Gynæcology" claims a foremost place. The records of clinical and pathological interest include "Atresia of the Conus Pulmonalis" by Dr. E. Cautley, and "Pulsation in the Second Left Intercostal Space" (by the right ventricle in a case of Mitral Stenosis) by Dr. C. Gibbes. Dr. McBride writes on Neuroses of the Throat and Dr. Sutherland on Neuroses in Childhood.

Amongst so much that is good it may seem fastidious to object to the article by Mr. Bland Bell on "The Causal Relation of Cocci to Rheumatism; but, whilst fully endorsing the author's opinion that clinical evidences of the causes of disease are often overlooked in these days of pathological research, we do not think that matters are likely to be improved by such loose statements as the following:—"There are many forms of intestinal absorption which we do not understand. If I had space I could quote cases, notably one of Landry's paralysis, which commenced almost exactly like an attack of acute 'rheumatic fever,' and was probably of intestinal origin." We also regard the inference that an attack of acute rheumatism was "coccal" because, ten days before, a swab from the tonsil showed "a few cocci, diplococci and micrococci in tetrads," as being quite unwarrantable. It is just such errors as these that demonstrate the need of those strict and persevering pathological investigations the value of which Mr. Bell rather discounts.

In Vol. XIII. there are contained an interesting paper on "Streptococcus Infections of the Lungs," by Dr. D. Lawson; a valuable contribution to "Post-mortem Appearances in the Drowned," by Dr. H. Littlejohn; and an account of a curious case of "Miliary Tuberculosis of the Conjunctiva," by Mr. G. A. Berry, along with many other contributions of importance. A useful section of the Journal is that dealing with "Recent Advances in Medical Science," although the paragraphs are mere transcriptions. The Book-Reviews are very carefully written, and are signed.

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Notes by the Way.

The Jenner Institute of Preventive Medicine.

We are very glad to record the signs of activity which are being manifested by the Jenner Institute of Preventive Medicine. In July last a new antitoxine department was inaugurated by Lord Lister in the presence of a distinguished gathering of scientific men from all parts of the country. Good wishes for the success of the institution were received from Professor Ehrlich of Frankfort, Professor Salomonsen of Copenhagen, Drs. Louis Martin and Dujardin-Baumetz of Paris, Professor Tavel of Berne, and other Continental authorities in the field of Bacteriology. The work of the new branch of the Jenner Institute consists in the preparation of the antitoxines which are in general therapeutic use, such as those for Tetanus and Diphtheria, and of Antistreptococcic serum, while opportunity will also be afforded for the study of other problems in the field of immunity against infectious disease.

The serum department of the Institute was formerly situated at Sudbury, near Harrow, but owing to the construction of the Great Central Railway through the old property it became necessary to obtain a new site. The new department is situated at Queensberry Lodge, near Elstree, Herts, and is presided over by Dr. George Dean. The buildings are on the summit of a small hill, and stand on a site of about 28 acres of land, which again are surrounded on all sides by open fields. The laboratories, which have been built after designs by Mr. Paul Waterhouse, are of the most modern type, with papyrolith floors, rounded corners, glazed adamant walls with a dado of white tiles, and large windows. There is a good gas and water supply.

The laboratories are designed in the form of separate blocks and work-rooms with the object of carrying out the processes involved in the most scientific manner. The manufacture of serums involves the keeping of a considerable number of horses, these being the animals generally used for the preparation of antitoxines. At the new institution the stables are particularly fine, consisting of several blocks, the majority of the horses being housed in roomy loose boxes.

It has also been practically resolved at a recent meeting of the governing body that the title of the Institute shall be changed from "The Jenner Institute" to "The Lister Institute of Preventive Medicine." The alteration is certainly a wise one, as the institution has suffered to a considerable extent in credit, owing to a confusion existing in the mind of the public between it and a private trading company for the supply of vaccine, which has a somewhat similar title. We very much hope that the change of name is an indication that the Jenner Institute, as we must still call it for the present, will undertake the preparation of vaccine among its other duties. The condition of things has for some time been very unsatisfactory as regards the supply of vaccine-lymph to practitioners of medicine who are not public vaccinators. It is to the latter alone that Government lymph is supplied, while the rest of the profession are obliged to depend for their lymph on the material manufactured by private commercial institutions, for the strength of which there is no guarantee. It is highly desirable that the Jenner Institute, with its high scientific reputation, and, if we may so call it, its semi-official position, should undertake the supply of so important a prophylactic.

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Sanatorium Treatment of Phthisis.

IN our present number will be found an interesting account, by Dr. Horton-Smith, of life in an open-air sanatorium for the treatment of consumption. Judging from this writer's experience the life in itself must be anything but an unpleasant one. Monotonous it may no doubt become after a time, but otherwise the patients have little to complain of. Of the value of the open-air treatment there can now be no doubt; but the question certainly arises whether this cannot be carried out as well at home as in a special institution. We have ourselves seen excellent results from open-air treatment as carried out at home, the patient spending most of the daytime in a shelter out of doors, and sleeping in a bedrom with the windows removed. There are, however, many dwellers in towns who have not gardens of their own in which to carry out this régime, and for them the provision of sanatoria is most important.

Further, it would seem advisable, wherever possible, for the patient to spend at least a short period of time in a sanatorium at the beginning of his treatment in order to learn the routine there observed, and the things to be done or left undone in order to gain the best results. After such a period of life under guidance, the rest of the treatment might well be carried out at home, whenever this was situated in the country.

The forced feeding, which formed at one time such a prominent feature in the sanatorium treatment of tuberculosis, appears now to be for the most part given up. Patients are encouraged to take a plentiful supply of nourishing food, but excessive amounts are not forced upon them as they once were, so as to create a loathing for it or even to excite vomiting. As it is, many patients put on a large amount of fat during their stay in a sanatorium, if they are doing at all well; and a fair amount of gentle exercise is necessary to aid them in the production of muscle and in the general nutrition of the tissues, while preventing to some extent the mere piling up of what the gentleman whose history is recorded in *The Bab Ballads* with "unpardonable vanity" preferred to speak of as "adipose deposit." Dr. Horton-Smith points out that the slow form of walking insisted upon for patients in sanatoria involves a much greater amount of exertion than would at first sight appear, the very deliberateness of the pace — slower than ordinary slow walking—causing increased muscular effort.

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Sanatoria for Advanced Cases.

THERE can be no doubt that from the point of view of the community at large the great danger resides in the infection spread by patients suffering from advanced stages of tuberculosis of the lungs. In such cases the sputum is often teeming with bacilli, and the spray of the cough, even apart from actual expectoration, or when this is received into proper vessels and duly destroyed, is a constant source of infection. As the end draws near, it is impossible for such a patient, however carefully he or she may have been instructed in hygienic measures, to carry out the rules of health effectually. In crowded homes among the poor, infection of the surrounding members of the household cannot fail to occur. Hence arises the necessity for providing Homes in which the sufferers may spend their last days, carefully tended so as to prevent spread of the disease, and supplied with the comforts needed to render their life as happy as it can be made. There might, it is true, as we are reminded in a recent letter of Dr. Alex. Robertson to the *Glasgow Herald*, be a difficulty in persuading some of the patients to enter such a hospital, the dislike of leaving home being a natural feeling under the circumstances. It has even been said that the so-called "*spes phthisica*" would keep them away from any institution, admission to which was felt to be a sign that the sufferer was beyond hope. But this would not necessarily be the case. The disease may be arrested in almost any stage; and as the most skilful treatment and nursing would be at the service of the sufferers, the very hope of cure might lead them to seek admission to the institution. As a matter of fact, we are inclined to think that the "*spes phthisica*" is more often read about than seen in the chronic cases of the malady. It is in the acute "galloping" cases that hope of recovery is at times so marked and so pathetic a feature.

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The Cost of Open-Air Treatment.

AN interesting article in the August number of *The Health Resort* calls attention to the financial side of the question of sanatorium treatment. The Assurance Societies in Germany have, it appears, taken up the question of providing sanatorium accommodation for poor consumptives at a reasonable cost. In one of their institutions a patient can be treated for a sum of about £1 a week, and as the average duration of stay may be put at four months, the cost per head works out at about £20—not an extravagant sum for the saving of a life worth perhaps £200 to the community. Nothing more is needed for the open-air treatment of phthisis than a hut of the simplest description, with a sufficiency of windows. A series of such huts can be put up at a very small cost; whereas the building of costly sanatoria, such as that to be erected under the scheme of the committee which is managing the plans for the King's sanatorium, comes to an enormous amount per bed. An illustration of a sanatorium on the hut-principle, of which it seems there are several already existing in this country, is given in the article alluded to. It seems to be along these lines that we may hope for the development of sanatoria under the care of our local authorities in the future.

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Treatment of Surgical Tuberculosis.

An important point is raised by Mr. Tubby in his paper in our present number as to the advisability, or even the legitimacy, of treating cases of tuberculosis affecting the bones or joints in hospital situated in large towns. The statistics which he brings forward seem to prove that the results thus obtained compare very unfavourably with those of country hospitals. It is certain that in London hospitals it is impossible to keep chronic cases in the wards for long periods of time owing to the great pressure which exists on the available bed-accommodation; and it is only after prolonged treatment that sufferers from surgical tubercle can expect relief. Nor does it seem to be the best arrangement to keep these cases in the close air of a large town, in which they languish rather than improve, after the first temporary amelioration due to improved food and attention has passed off. Country air is most needful for these little patients—for it is among children that the majority of these forms of tubercular disease are met with—and some arrangement such as Mr. Tubby suggests, whereby they could all come under the consideration of a central council, and have their needs systematically investigated, is devoutly to be wished. At the same time there are difficulties in the way of the scheme. The arrangements as to hospitals in general—their relation to the public and to one another—are at present chaotic, and much previous organisation will have to take place in many directions. We can hardly start by establishing a central council to supervise a single form of disease and leave all the rest unorganised.

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Report of the Cancer Research Fund.

We cannot say that the foundation of a central institution for the investigation of cancer filled us with any enthusiastic expectations that the cause of the disease was immediately going to be discovered. We shall still be quite as hopeful of hearing that some individual researcher working on his own account has revealed the mystery, as that the solution has been found in the laboratories specially endowed for the purpose of this quest. At the same time we are glad to see that the directors of the latter are apparently proceeding on wise lines, in inviting the different hospitals and schools to collaborate by furnishing reports of all cases of malignant disease which come under treatment or are submitted to post-mortem examination. Much valuable material will, no doubt, be collected in this way. The recently-issued report of the Superintendent of the Research Fund indicates that investigation of cancer as it occurs among the lower animals will occupy a foremost place at the outset of the enquiry. In this direction lie the best hopes of elucidating the causation of the disease, as experimental inoculation is impossible on human subjects, whereas it is by experiment that practically all the greatest medical discoveries of modern times have been made. The remarkable results obtained in the treatment of rodent ulcer by means of X-rays form one of the most gratifying features connected with the subject of tumours, but they cannot be said at present to throw any light on the nature of the process. No success seems to have attended the attempt to cure internal growths by this means, and in spite of the sensational headings used to advertise some of the evening papers, there cannot be said to be yet any "cure for cancer" in the ordinary acceptation of the term.

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Feeble-minded Children.

FEW more difficult problems come before the physician than those connected with children who are the subjects of mental defects which lead to their classification as imbecile or feeble-minded. It is difficult to know what exact advice to give as to their upbringing, and even more hazardous in many instances to venture an opinion as to the probability of future improvement. Only one who has had a large experience of similar cases can form a judgment as to the likelihood of mental progress being made under suitable guidance and instruction, or whether the case is one insusceptible of improvement even under the most skilful system of education. In our present number Dr. Tredgold gives a valuable account of the different forms of mental defect, hereditary and acquired, with graphic illustrations of the principal types. It is impossible not to regard with feelings approaching to abhorrence some of the grosser forms of hereditary idiocy. On the other hand many of those who have acquired the mental defect are of ordinary or even pleasing personal appearance. It is difficult to believe—yet it is undoubtedly the case—that in these acquired forms, presenting features of average intelligence, the outlook is usually worse than in the ill-developed subjects of hereditary defect. Much may be done for the milder forms of the latter condition. In their early years they must perforce be left to be brought up by their parents, but later on it is almost necessary for their advancement that they should be cared for in some special institution, where skilled attention can be paid them. Few perhaps improve so much that they can ever leave the shelter of the home and take care of themselves in the world, but within the institution they may become useful individuals and help at least to earn their own livelihood.

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Practical Notes.

THE USE OF COLLOIDAL SILVER IN INFECTIOUS DISEASES.—Dr. Netter (*Journal de Médecine*, 1903, Vol. XV., p. 14) enumerates a number of infectious diseases that have been promptly relieved by the use of colloidal silver or collargol, which was introduced as a therapeutic agent in 1897 by Credé. It is an allotropic form of silver which is soluble in water and in the secretions of the body. Colloidal silver is used chiefly for external applications in the form of a salve, 15 to 100 (*Unguentum Credé*), and in intravenous injections in a solution of 1 to 200. The amount of salve used in an application is 15 to 45 grains, according to whether the patient is a child, a youth, or an adult. For intravenous injections the amount varies from one half to five-sixths of a grain. The use of collargol has grown widely, and many physicians report remarkable cures in many different diseases. Wenchebach cites two cases of severe infectious endocarditis that were cured by means of the injections, while Klotz obtained wonderful results in a septic endocarditis. The author reports ten different kinds of cases in which he had excellent effects from this remedy, among which was a case of pneumonia in which, after intravenous injections, the fever dropped immediately and disappeared entirely in three days. A suppurating cerebrospinal meningitis was modified as suddenly, apyrexia ensuing the day after the injections, and a rapid convalescence. The results were no less satisfactory in a severe scarlatina, two toxic diphtheritic anginas, and in three adynamic typhoids. The last observation was of a medico-surgical nature. A physician seized with pyæmia found almost instant relief after a single application of collargol. Both the patient and the nurse who made the application noticed a distinct metallic taste. The return of strength, appetite, and ability to move came the following day. It is not certain that collargol acts antiseptically. Perhaps there is a neutralisation of the toxins which produce such rapid changes in the body, or perhaps there is stimulation of the defensive power of the organism, or a catalytic action. (*Therapeutics.*)

PAINFUL AFFECTIONS OF THE FEET IN NURSES.—R. W. Lovett (*American Medicine,* July 4, 1903) has investigated the painful conditions of the feet common among nurses, and comes to the following conclusions:—

1. It has not been possible to tell with any certainty by examination whether or not the feet of an individual are likely to give trouble. The only reliable information obtained in these cases was given by the imprints seen through glass. A foot with a well-distributed pressure-area seemed rather less likely to give trouble than one resting on two islands; the degree of pronation, the condition of the circulation, the relative weight of the nurse, and the dorsal flexibility of the foot all proving of little or no value as elements in prognosis. A flat foot may be perfectly serviceable as may also a severely pronated one, while an apparently well-balanced foot may become painful.

2. The factors in causing the trouble among the nurses were to be sought rather in the general conditions than in any especial conformation of the foot. It followed in many cases, illness and other conditions causing muscular debility. It occurred in most cases from two to three months after entrance, and it began most often in the early spring when the nurses had been indoors all winter, and least often in the fall.

3. The trouble was caused by a rolling in of the foot and a shifting inward of its weight-bearing areas and not in any case observed by a breaking down or even lowering of the arch.

4. Although proof by figures was lacking it was probable that the amount of trouble had been decidedly less than it would have been without the use of a proper boot.

By way of treatment, in milder cases, felt pads were used to support the arch of the foot, and the nurse was ordered to soak the feet in hot water at night for ten minutes, and then to shower the feet with cold water and to bandage the feet and legs with flannel for the night. The majority of early cases yielded to this treatment. If, however, rapid improvement did not come on in two or three days, or if the first symptoms were severe, a metal support to the arch of the foot was at once put in and measures to stimulate the circulation were used. In some cases plates relieved the symptoms at once and were worn for a few weeks or months, generally to be given up later.

In the severest cases all sorts of expedients were tried to cure the pain while the nurse continued her work. The most efficient support of all is to apply a felt pad to the instep while the foot is held inverted, by fastening the pad to the bare foot by several straps of adhesive plaster which pass from the outer border of the foot under the foot and up under the inner side of the arch over the top of the foot to the ankle. These straps overlie each other, and are covered by a heavy cotton bandage. This dressing must be renewed every day or two. This dressing, which is distinctly more supporting than any metal plate, in some cases carried nurses over a painful attack while on duty, but more often it failed in the severest cases. In these severest cases hot-air baths, massage, and stretching of the calf-muscles were tried with no brilliant success. They helped the condition, but the writer cannot recall a severe case in which any one of these latter measures has made any serious difference.

If a proper fitting metal plate fails to relieve symptoms within two or three days, the sooner the nurse is laid off the better, and the shorter her period of incapacity. When she is laid off she should not be allowed to walk, but as she convalesces she may be encouraged to take out-of-door walks. Several times it was demonstrated that it was unwise to allow nurses to go on duty under other conditions. Strychnine in fairly large doses was used in a number of cases in the hope of improving the muscular tone when trouble was threatening, but the writer failed to see striking instances of its benefit.

VERONAL AS A HYPNOTIC.—Lotsch (*Fortschritte der Medicin*, July 1, 1903) writes of the good effects of veronal in cases of sleeplessness. He has tried it in a number of different conditions, among them cases of cardiac failure and phthisis, with the best results. The dose is approximately 4 to 15 grains. Small doses should be administered at first, until the effect on the individual patient is known. Veronal is not an analgesic, but in cases in which pain is absent it procures, as a rule, eight hours of refreshing sleep. It is without ill effects, when given in the amounts mentioned, though in larger doses it may be followed by an erythematous eruption.

ABORTIVE TREATMENT OF BOILS.—Gallois (*Therap. der Gegenwart*, 1903, II.) advises for this purpose that the boil should be painted with a concentrated solution of iodine in acetone. This solvent takes up four times as much iodine as alcohol, and its use on a boil is followed by the formation of a brown scab, under which the inflammation subsides. The solution should be kept for some weeks before it is used.

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THE PRACTITIONER.

OCTOBER, 1903.

TWO LECTURES
ON
THE DIAGNOSIS OF HYSTERIA.[1]

BY PURVES STEWART, M.A., M.D., M.R.C.P.,
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(With Plate XVII.)

LECTURE I.

GENTLEMEN,—When we talk as physicians of Hysteria we all have a fairly clear general idea of what we mean. And yet if we are asked the question "What is hysteria?" we find it somewhat difficult to express our definition in words.

At the outset we should recognise that hysteria is a real disease, as real as small-pox or cancer, and that it has a physical basis, probably of a chemical nature, although this is yet very imperfectly understood. We must draw a sharp distinction between a hysterical patient and a person who is deliberately shamming or malingering. The imitation of other diseases which is so often seen in hysteria—*neuromimesis*—is not a voluntary imitation. On the contrary, the various paralyses and pains from which so many hysterics suffer are as real to the patient as if they were due to gross organic causes.

All modern observers agree that the site of the disease is in the nervous system and various hypotheses—vasomotor, nutritional and others—have been suggested to account for it. I believe that hysteria is a disease of nerve-metabolism affecting not only the highest or psychical centres, but implicating the whole nervous system in varying degrees, and producing symptoms corresponding with the parts affected.

Some hysterical symptoms are psychical, due to affection of the highest centres; others, sensory, motor and reflex, are the result of affection of the sensori-motor cerebro-spinal paths; others, again—visceral, vasomotor, etc.—are referable to the sympathetic nervous system.

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[1] Delivered at the Medical Graduates' College and Polyclinic.

Such disordered nerve-metabolism may show itself in three ways:—

1. By excessive activity.
2. By diminished activity.
3. By perverted activity.

The basis of hysteria, as of all diseases, bodily or mental, is physical in nature. In many instances this physical basis is a toxic one, the poison either being produced within the body or introduced from outside. In others, gross anatomical changes may induce hysterical symptoms. Just as gastric symptoms may arise not only from faulty chemistry within the stomach, but from gross organic disease in its neighbourhood, so also certain cases of serious organic disease, *e.g.*, disseminated sclerosis and some cerebral tumours, may produce at first symptoms which are indistinguishable from hysteria—which, in fact, are hysterical. And in such cases we must try to detect the underlying organic disease and to differentiate its symptoms from those of the superadded hysteria. Hysteria, therefore, may coëxist with organic disease. In practice, however, we generally restrict the term hysteria to those cases where no organic disease is present.

In this and the succeeding lecture I propose to remind you very briefly of some of the most characteristic diagnostic symptoms of hysteria and to point out in what respects they differ, if they differ at all, from the analogous symptoms produced by gross organic lesions.

Time does not permit me to discuss the various predisposing and exciting causes of hysteria, such as sex, age, nervous heredity, physical and mental shock, etc., or its frequency in certain races, especially among the Latin races and the Jews.

For purposes of convenience we shall consider the symptoms in four groups:—

A. Psychical.
B. Sensory.
C. Motor.
D. Visceral, Vascular, etc.

In each of these groups we may find excess, diminution, or perversion of the normal nervous processes.

A. PSYCHICAL SYMPTOMS.

Psychical symptoms are invariably present in hysteria to a greater or less degree. The most outstanding feature is *deficiency of inhibition.* The patient reacts too readily to stimuli or suggestions, whether originating in the outside world or within her own body. One of the most striking instances of this is found in the phenomena of hypnotic suggestion, whereby a peculiar form of temporary hysteria—the hypnotic trance—is artificially induced by suggestion and can be made suddenly to disappear by the same means. This disappearance of symptoms during or after hypnosis is sometimes taken advantage of in the treatment of hysteria by hypnotic suggestion. But it is open to the objection that instead of strengthening the patient's feeble inhibition, it utilises that fault. Yet if the result be that the patient has a hysterical suppression of her hysterical symptoms (on the mathematical principle that $- \times - = +$) it is, to some extent, a benefit. But results obtained by this plan are less likely to be permanent than when we strengthen the patient's inhibition or self-control by positive measures.

Deficient inhibition being the keynote of the hysterical "*psyche*," we find, accordingly, that the patient is excessively emotional and changeable in disposition, often excitable, and perhaps passionate. She giggles or cries on slight provocation, and one of the most familiar forms of hysterical "fit" consists mainly in alternate loud laughter and crying. Her will-power is feeble, she is swayed by passing whims; and this want of self-reliance leads to another very characteristic symptom—the craving for sympathy. If that sympathy be shown, as is so often the case, to an injudicious extent by the patient's relatives and friends, her recovery may be thereby indefinitely delayed. Hence it is of supreme importance, in many cases, to remove the hysterical patient completely from her old surroundings, and to isolate her until the vicious circle is broken.

I show you a photograph (Fig. 1) of a woman of 37, who for over 14 years had lain on a bed of sickness unable to move her legs, a case of hysterical paraplegia. But she was cherished by the sympathy of a devoted mother and of various benevolent lady-visitors, anxious to soothe her dying moments. She was taken to hospital, and as a result of six weeks of isolation and massage, she recovered the power of walking, as you will see from this other photograph (Fig. 2). And this successful result was due quite as much to the isolation as to the other measures, such as massage, special diet, etc.

It is convenient to mention here, in connection with the psychical symptoms, the *hysterical affection of speech.* Sometimes there is excessive volubility—a *diarrhœa verborum.* In other cases we find the reverse condition of hysterical mutism where the patient is absolutely dumb. In less severe cases it is not uncommon to find merely loss of voice, or hysterical, aphonia—where the patient can only whisper her tale of woe, breathing it softly into the ear of a sympathetic listener. Hysterical aphonia has characteristic laryngoscopic appearances, in the form of adductor paralysis of the cords, with which we are all familiar.

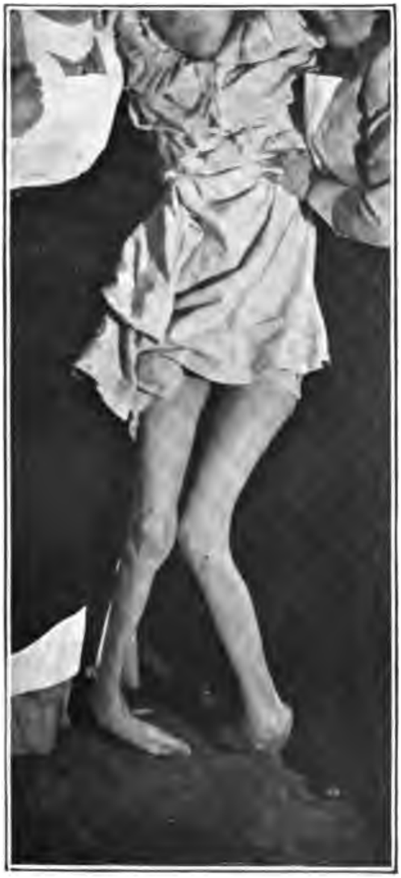
Patients with such mutism or aphonia sometimes suddenly recover their voice when the dominant obsession is loosened, *e.g.*, by administration of chloroform (by the physician), or of an excess of alcohol (by the patient), or by some sudden shock, physical or mental. Intra-laryngeal faradism cures many cases of hysterical aphonia at once.

Articulation in hysterical patients may be affected in all sorts of curious fashions. I have seen several patients who drew a breath between each separate syllable, *e.g.* "hos—pi—tal." Others interpolate curious barking, grunting, or snorting noises amongst their words. One lady whom I met was afflicted in this way to such an extent that new neighbours in an adjoining house thought the noise was made by a sick dog, and sent in a suggestion that the animal should be humanely put out of its pain. And yet this same lady could recite long dramatic passages of poetry and prose, though in ordinary conversation, or even when not talking, her bark made her society a somewhat mixed pleasure.

Stammering in its different varieties is not uncommon in hysterical patients. Unlike ordinary stammering which comes on in childhood, hysterical stammering may develop suddenly in adult life. Thus in a hospital nurse of 33, who stood up

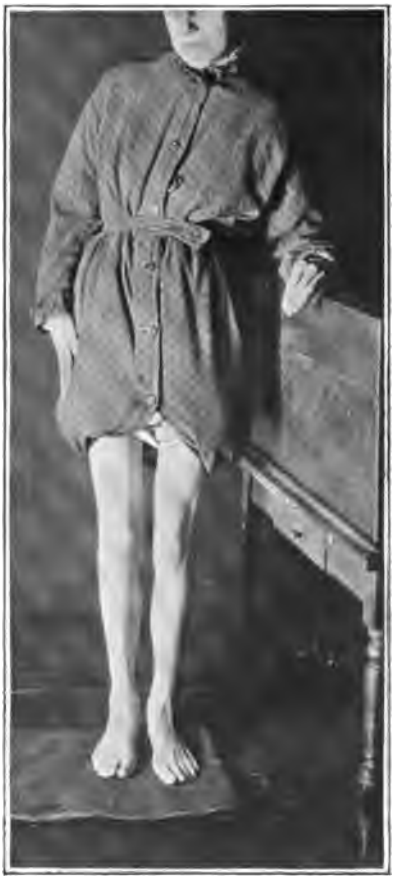
Plate XVII.

Fig. 1.



Case of Hysterical Paraplegia of fourteen years' duration.

Fig. 2.



The same patient as in Fig. 1, after six weeks' treatment.

suddenly and knocked her head against a mantel-shelf, severe hysterical hemiplegia came on next day, and stammering some six weeks later, lasting for several months.

B. SENSORY SYMPTOMS.

These are of greater diagnostic importance than is commonly realised. *Pain* of some sort occurs in nearly every case of hysteria. Unlike ordinary pains, which are generally caused by some peripheral irritation, hysterical pains are entirely central in origin—*psychalgia*—and should really be classed as hallucinations. But in every case we must be careful to see and examine the painful spot, and to exclude peripheral irritation, before labelling any pain as hysterical.

Hysterical pains may be referred to any part of the body. But they are specially common in certain situations, *e.g.*, hysterical headache may be of the "clavus" type, which is a boring pain localised to one small spot on the skull. Occipital headache is particularly common, so is hemicrania. Pains in the spine may simulate organic disease. Pain in the breast—mastodynia—or in the joints may lead to difficulties in diagnosis. It is only by careful local and general examination, by the presence of other hysterical stigmata, and by the absence of signs of structural disease, that we can avoid errors. Cases have been known in which amputations have been performed for hysterical joint-pains. Only last summer I saw a young lady who had already had one toe removed by a surgeon, but the pain recurred, and the case was undoubtedly hysterical.

Closely related to these pains are the areas of hysterical hyperæsthesia, and especially of hyperalgesia or excessive tenderness. Universal hyperæsthesia is rare. I have met with several cases of hemi-hyperæsthesia. More usually this excessive tenderness is circumscribed in small areas—little islands of skin or subjacent tissues, on the head, trunk, or limbs. Sometimes the tenderness is cutaneous and elicited by gentle stroking of the skin; sometimes it is subcutaneous, and only elicited on deeper pressure.

Such tender points are chiefly situated in the vertebral, infra-mammary, epigastric and inguinal regions, and except when mesial, are more frequently left-sided (except in left-handed people in whom they are more commonly right-sided). Tender points are less common on the head, and rarest of all on the limbs.

Of all the tender spots, that in the left inguinal region is perhaps the commonest. From some supposed connection with the ovary, it has been called "ovarian" tenderness, but the symptom is as frequent in male hysterics as in females, so that the term is a misnomer. Moreover, in this connection, Steinhausen[1] examined 500 healthy soldiers—males, not Amazons—and found that in no less than 88 per cent. brisk pressure in the inguinal region on either side produced a reaction of some sort, the phenomena being either sensory (unpleasant tickling or pain), motor (hardening of abdominal muscles, various reflex and protective movements), psychical, or vasomotor and sympathetic (dilatation of pupils). And yet there was not a single ovary amongst them.

These tender points may be associated not merely with pain, but with so much disturbance as to be actually *hysterogenic.* This does not mean that they induce hysteria—the hysteria is already present. It means that pressure on such a spot induces a hysterical fit or paroxysm. The best-known hysterogenic area is in the left inguinal region, but such areas may be anywhere. I know of one patient who had a hysterogenic spot in one axilla, and who wore a sort of truss over it to prevent accidental pressure.

Sometimes deeper pressure on the hysterogenic spot or elsewhere may arrest a hysterical fit when in progress. Areas, pressure on which causes cessation of the paroxysms, are called *hysterofrematic.* The inguinal region is the best known of such areas. I think it was Mr. Lawson Tait who observed that brisk tugging at the pudendal hair had a vigorous arresting effect on hysterical fits. Personally I have never tried this. Strong faradism over the inguinal region will stop most hysterical fits.

Hysterical hyperæsthesia may affect the special senses as well as the skin and muscular sensibility, so that we may get hyper-sensitiveness of smell, vision, hearing, or taste. This is less common than loss or diminution of special senses, to which we shall refer later. I have at present under my care a patient who could not tolerate bright light, especially if the

[1] *Steinhausen*: "Ueber die physiologische Grundlage der hysterischen Ovarie." (Deutsche Zeitsch. f. Nervenheilk., XIX., p. 369.)

room had a blue wall-paper. He preferred to stay in a darkened chamber; or if the blinds were up, he shaded his eyes with his hand. Now, after some weeks of treatment, he has completely lost his photophobia.

In rare cases an actual enlargement of the visual field has been observed, generally in one eye only. Thus, in a soldier whom I examined, with hysterical wry-neck and anæsthesia of one side of the body, the visual field on the non-anæsthetic side, when measured with the perimeter, was much larger than in a normal individual.

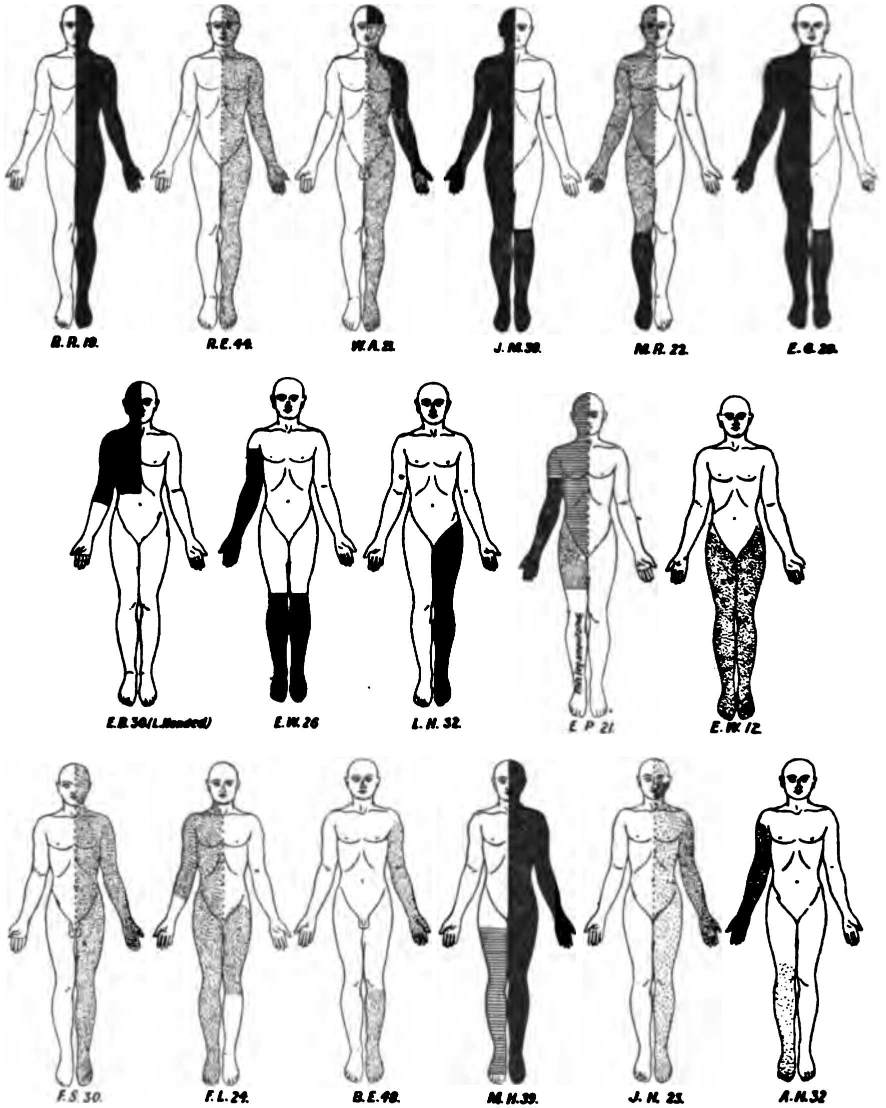
Hysterical anæsthesia is extremely common, and is of the greatest diagnostic value. A degree of anæsthesia exists, I am convinced, in the overwhelming majority of hysterics. I have looked through my notes of 62 cases and find that anæsthesia was present in 49 and absent only in 12. The remaining one case of the series had unilateral hyperæsthesia.

Hysterical anæsthesia is often unnoticed by the patient herself, and only discovered on examination by the physician. Sometimes, however, the patient complains of actual numbness, and this is chiefly in cases where the affected limb has motor paralysis as well, so that her attention is called to it.

Janet, of Paris, showed an ingenious method of demonstrating that in some cases of hysterical anæsthesia, sensory impulses really reach the brain-centres, though the patient does not consciously perceive them. Take a case of complete hemianæsthesia. Make the patient shut her eyes, and tell her to say "Yes" each time she feels a touch or prick, and to say "No" when she does not feel it. In certain cases the patient not only says "Yes" every time she is touched on the normal side, but also says "No" every time she is touched upon the anæsthetic side!

Cutaneous anæsthesia in hysteria may be complete or partial in degree, or it may be dissociated. Diminution or loss of painful sensations is even commoner than tactile anæsthesia. Many of the mediæval witches were simply hysterics. Their hysterical analgesia was usually demonstrated by sticking pins into them, and if an analgesic area was discovered, they were promptly thrown into the nearest pond or stream. If they sank and were drowned, their innocence was established; but if they floated, it was additional evidence of guilt.

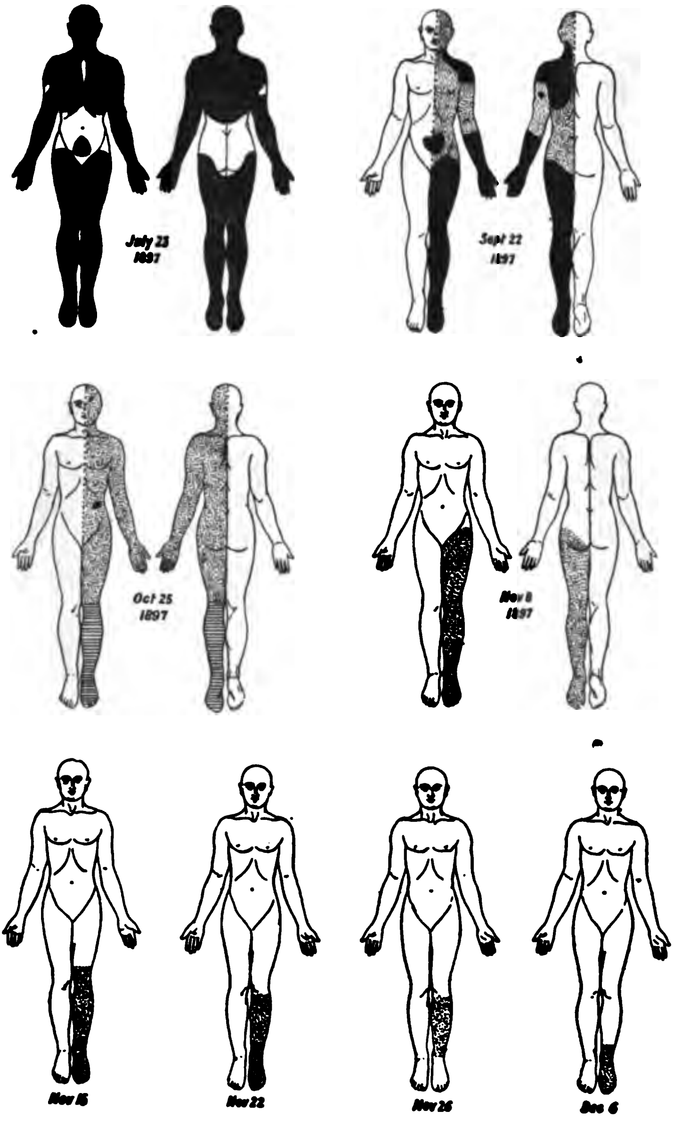
Fig. 3.



Various Types of Functional Anæsthesia.

But whatever be its degree of intensity, hysterical anæsthesia never maps out an individual nerve-area such as that of the radial, median, ulnar, or external popliteal. Its commonest distribution is a *hemianæsthesia* (26 out of 49 cases) which is mostly left-sided (except in left-handed patients).

Fig. 4.

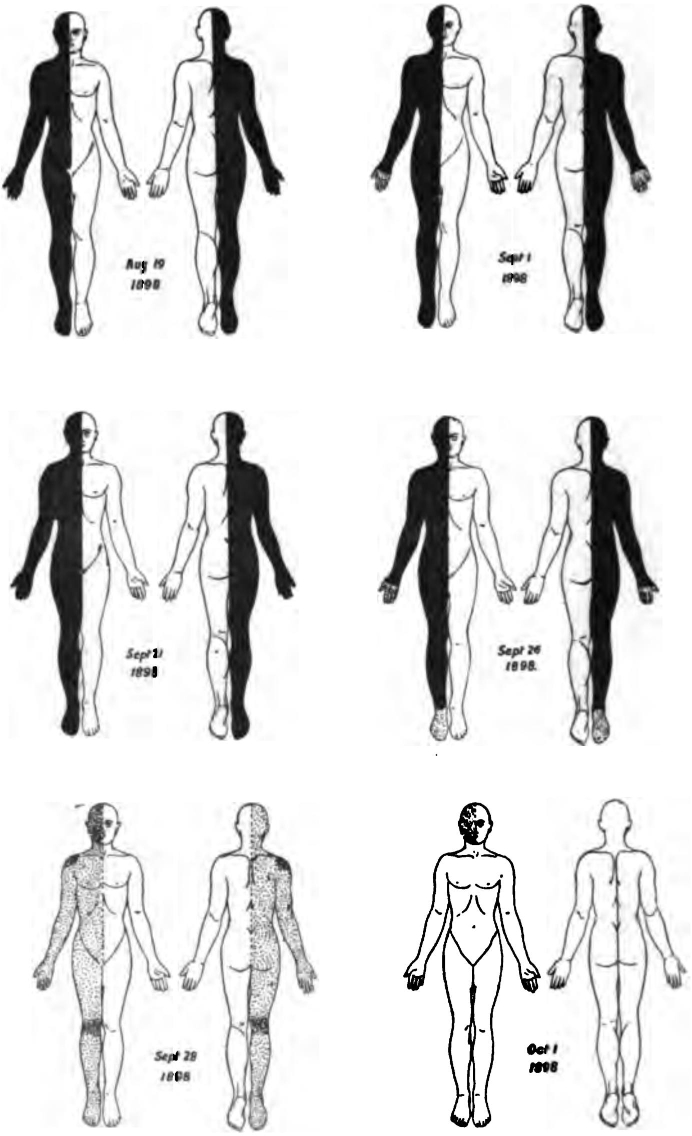


Case of Functional Paralysis.

S. S., *æt.* 17.

Hysterical hemianæsthesia, including the accessible mucous membranes of the eye, nose, mouth, pharynx, vagina, and

Fig. 5.



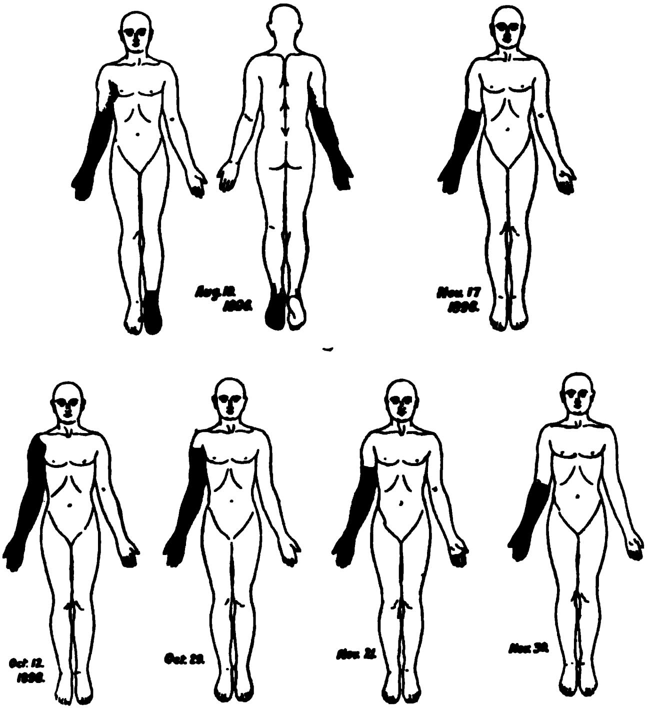
Case of Functional Hemiplegia.
E. L., æt. 33.

rectum, is usually accurately bounded by the middle line; but not always. It may either extend farther over and encroach on the non-anæsthetic side, or it may leave certain areas with normal sensation, even on its own side, especially the head and the genitals, as you may see from some of these charts (Figs. 3, 4, and 5).

Bilateral universal anæsthesia is rare. We generally find one or more islands of normal sensation, or even of hypersensitiveness, somewhere or other.

In many cases the anæsthesia, though unilateral, is more marked on the face or limbs than on the trunk. Or it may affect special levels of a limb (knee, elbow, or shoulder), or it may stop abruptly at some horizontal line (shoe, sock, stocking, mitten, glove, sleeve). This "*segmental*" *anæsthesia* sometimes occurs in association with hemianæsthesia (10 out

Fig. 6.



Case of Functional Paralysis.

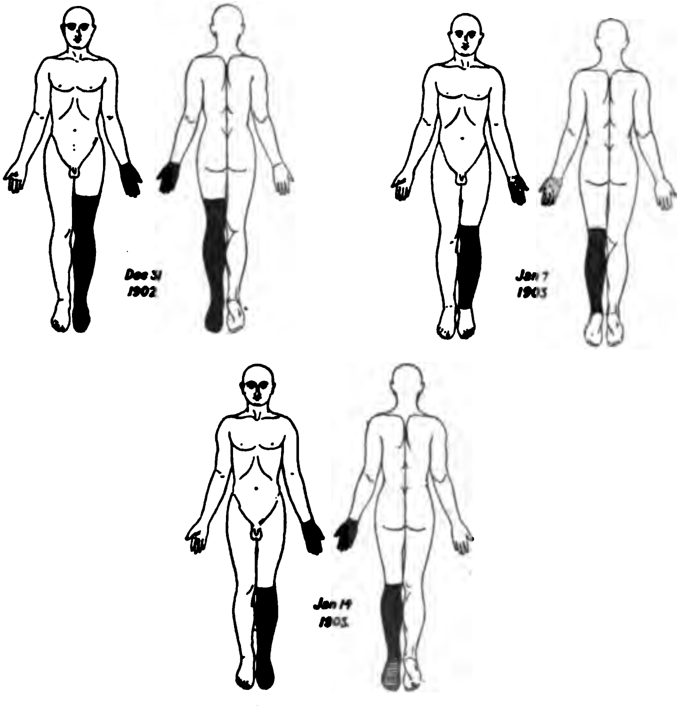
C. D. (*L. Handed*).

of 49 cases) or by itself (12 out of 49) on one or both sides. (*See* Figs. 3, 6, 7, and 8.)

The mode of onset and disappearance of hysterical anæsthesia is interesting. Sometimes the anæsthesia comes on gradually, and the patient is unconscious of the defect. In other cases it occurs suddenly, especially after a hysterical fit, and the patient is then more likely to notice her "numbness."

We seldom have the chance of watching the onset of hysterical anæsthesia, but we may often study its mode of disappearance, and Figs. 4 to 8 show charts of different cases of hysterical anæsthesia in various stages of recovery. Unlike organic anæsthesia, which, if it recovers, fades gradually all over the recovering area, hysterical hemianæsthesia may suddenly fade to segmental (*see* Fig. 4), and segmental anæsthesia recedes by jumps from a higher to a lower level, bounded usually

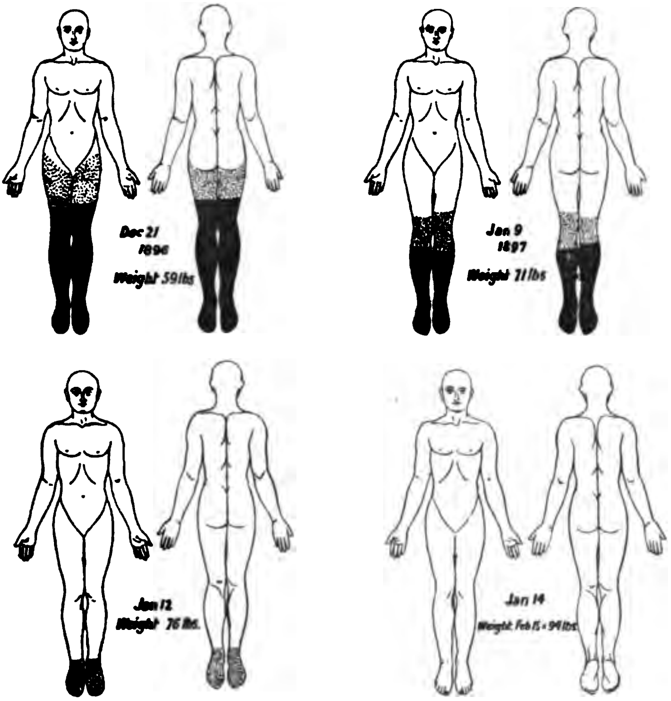
Fig. 7.



Case of Functional Hemiplegia.
J. L.

by an "amputation line" drawn transversely across the limb. Sometimes it relapses temporarily to its old level before resuming its progress towards recovery (*see* Figs. 5 and 7). More rarely it clears up first at the periphery.

Fig. 8.



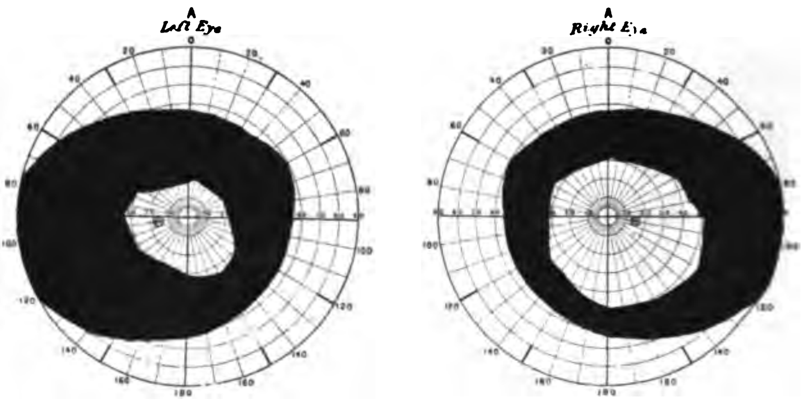
Case of Functional Paraplegia.
E. A., æt. 19.

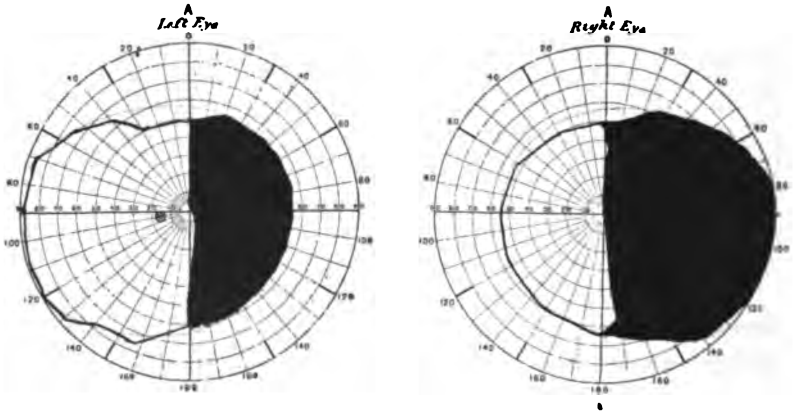
Anæsthesia of Special Senses.—Most cases of hysterical anæsthesia have also got diminution or loss of the special senses—smell, vision, taste and hearing—generally unilateral and on the same side as the cutaneous anæsthesia, rarely on the opposite side. This combination of unilateral affection of special senses and of cutaneous sensation is pathognomonic of hysteria. It does not occur in organic disease.

Moreover, the affection of vision is not a hemianopia such as we often get in organic hemiplegia. It is a concentric contraction of the whole visual field, as you will see from these perimetric charts (Fig. 9). And this is more marked in one eye than in the other—"crossed amblyopia"—the smaller field being on the hemianæsthetic side.

Fig. 9.

Crossed Amblyopia (Functional).





Right Homonymous Hemianopia (Organic).

The colour-fields are also narrowed, but in a different order to that which occurs in organic optic atrophy. In hysteria the blue diminishes first and the red last of all, whereas in organic atrophy the red field is usually first affected.

Sometimes we have actual hysterical blindness or amaurosis in one eye, and yet by means of prisms we can often produce a diplopia, which in organic cases would be impossible. In very rare cases complete bilateral hysterical blindness has been observed.

Time does not permit me to discuss the other peculiarities of hysterical eye-affections and of hysterical loss of smell, taste, and hearing, but I may mention, in passing, that complete bilateral loss of taste, as Dr. Hughlings Jackson has pointed out, is practically always hysterical.

Hysterical Paræsthesia, or perversion of sensation.—In some cases of hysteria a touch on one limb or one side of the body is felt by the patient at the corresponding spot on the opposite side—*allochiria* (Obersteiner). In another variety, of which I have seen an example, a touch on the radial border of the limb was felt on the ulnar, and *vice versâ*. *Haphalgesia* (Pitres) is the term used when intense pain is caused by touching the patient with certain substances, such as metals, which should only cause a tactile sensation.

Paræsthesia may also affect the special senses, as for example in *monocular diplopia or polyopia*, which is always hysterical, and in *micropsia*, when everything looks very minute, or *macropsia*, when surrounding objects seem gigantic.

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THE RESULTS OF ORGANOTHERAPY IN ADDISON'S DISEASE.

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ORGANOTHERAPY in Addison's disease has just now entered on its second decade. A space of time has, therefore, elapsed sufficient to judge, to some extent, of the validity of its claims and its future prospects. That suprarenal feeding in Addison's Disease has by no means given results comparable in brilliance with those obtained by thyroid feeding in myxœdema and cretinism is of course well known. For the rest we prefer first to submit the facts, so far as they are at present known and recorded, before making any further statement.

It is not claimed that every published case of Addison's Disease treated by organotherapy is to be found in the collection here given, but it is hoped that but few of the more important records occurring in English, French, and German literature have been omitted, and the number (97) is probably sufficient to allow of a few conclusions for the time being. Though my inferences should not commend themselves to all, I hope at least to have furnished data, as reliable as I could, which may prove of some service to abler reasoners.

Of previous collections undertaken with the same main object perhaps the best-known is Kinnicutt's (48 cases).[1] I have not, however, availed myself of the excellent work of others in this direction, since my aim, though roughly the same in general plan, is not so in detail. I am, therefore, alone responsible for the accuracy or otherwise of every reference and summary here given, with one exception, which is indicated.

In addition to cases collected from the literature, I have been enabled by the kindness of colleagues to include six unpublished cases of importance, and also in some of the cases classed in Groups III. and IV. (*vide infra*) to bring the after-histories more or less up to date. Cases thus enquired of are marked with an asterisk, and the additional information (if any) is given in a subsequent section. I desire here especially to acknowledge my obligations to Professor William Osler, Dr. George Oliver, Dr. John Hay, Dr. H. D. Rolleston, Professors

James Anders, Chas. G. Stockton and Ralph Stockman, Dr. Lloyd Jones, Dr. A. E. Sansom, Dr. C. W. Suckling, Dr. D. N. W. Murray, Professor Anderodias, of Bordeaux, and Dr. C. R. Box for their courteous replies to my queries, and their kindness in furnishing me, where possible, with valuable and unpublished information as to the subsequent progress of their respective cases.

The cases have been arranged in four groups:—

Class I.—Cases in which alarming or fatal results were presumably or possibly due to the treatment;

Class II.—Cases uninfluenced by, or deriving but doubtful benefit from, the organotherapy;

Class III.—Cases in which marked improvement coïncided with the treatment; and

Class IV.—Cases in which permanent benefit (? cures) accrued apparently as a result of the suprarenal feeding.

No case is included under Group IV. which has not been under observation for at least a year.

The effects of the treatment upon the three cardinal sets of symptoms of Addison's Disease have been tabulated. These cardinal symptoms have been taken to be:—(1) The circulatory feebleness; (2) general symptoms, such as asthenia, gastro-intestinal troubles, wasting and lumbar pain; and (3) pigmentation. Since the mode of employment of the adrenal substance in each individual case is of great interest and moment, this has also been noted where recorded. Finally, the ultimate result, as stated by the author referred to, is given.

In Classes III. and IV., where improvement has resulted presumably from the organotherapy, an additional column has been inserted, at the head of which will be found: "Other Treatment Used." The information entered in this column is obviously of importance when estimating results and drawing conclusions. As has been stated, additional information has also been obtained concerning some of the cases in these groups.

The list might have been extended had I cared to make use of several secondhand references which I have been unable to trace, but this would have been unwise.*

The number of cases here recorded is 97.

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* *E.g.*, Dupaigne (*op. cit.*) attributes Dr. George Oliver's two cases to Dr. Thomas Oliver, and gives as his reference *Internat. Clinics*, 1894. The same error is repeated by another French writer.

GROUP I.

Alarming or Fatal Results presumably or possibly due to the Treatment.

No. of Case.	Reference.	How employed.	Effect on Pulse.	Effect on General Symptoms.	Effect on Pigmentation.	Ultimate Result.
1	Bra. *La Thérapeutique des Tissus*, p. 528. (Reported by M. Augagneur.) (Child of 14.)	Suprarenals of dog grafted into cellular tissue of abdomen.	—	—	—	Death on third day preceded by fever and coma.
2 & 3 (Two cases)	Jaboulay. *Lyon Médical*, Mars 21, 1897.	Grafts of suprarenals of dog on each side of abdomen subcutaneously under local anæsthesia.	—	—	—	Both patients died within 24 hours, with symptoms of hyperpyrexia and prostration identical in each.
4 & 5 (Two cases)	Affleck. *Lancet*, Dec. 31, 1898.	Suprarenal gland-extract -	—	Alarming collapse. One of the cases began to improve markedly when extract was stopped.	None - -	Drug had to be discontinued. One case ultimately fatal.
6	Destot. *Lyon Médical*, Nov. 27, 1898. (*Soc. Nat. de Méd. de Lyon.*)	Injections of aqueous suprarenal juice gradually increased.	—	Gastro - intestinal symptoms made considerably worse.	Diminished - -	Net result unfavourable. Treatment discontinued.
7	Rendu. *Gazette des Hôpitaux*, Février 28, 1899.	One suprarenal capsule of calf by mouth daily.	—	—	—	Notable diminution in quantity of urine. Sudden death 10 days after commencement of treatment.

Some very unfavourable cases have also been reported in Italian literature, but I am unacquainted with the language. Pitres has also reported two cases where the treatment caused a manifest aggravation of the general state; but I found the reference given incorrect, and have been unable to track the right one.

GROUP II.

Cases deriving no, or but doubtful, Benefit from the Treatment.

No. of Case.	Reference.	How employed.	Effect on Pulse.	Effect on General Symptoms.	Effect on Pigmentation.	Ultimate Result.
8	Abelous, Charrin, et Langlois. *Archives de Phys.*, 1892, p. 724.	Aqueous extract of suprarenals of horse and dog by hypodermic injection.	—	A slight diuresis noticed	—	Indefinite. Treatment not pushed.
9	Chauffard. *La Semaine Méd.* Février 14, 1894.	Suprarenal juice injected deeply into buttocks from 23 to 31 March. Great pain, but no local lesion produced.	—	Perhaps a slight augmentation in strength.	—	Injections gave great pain, lasting some hours. Did not avert an early fatal termination.
10	Bra (communicated by Dr. Langlois). *La Thérapeutique des Tissus*, p. 357. Also Mahé, *Thèse de Paris*, 1894, p. 51.	Injections of suprarenal liquid every other day.	Not appreciable	Doubtful slight increase in strength.	Not appreciable	Punctures very painful. Patient refused further treatment.
11	G. Lyon. *Revue de Thérapeutique*, Juillet 1, 1895. (Two or three cases.)	Injections of capsular extract prepared after Langlois' method and also D'Arsonval's.	—	(These patients had advanced pulmonary tuberculosis.) Not appreciable.	—	Patients of G. See. Lyon observed no benefit.
12	Bradshaw. *Lancet*, Oct. 31, 1896. (Male, 31.)	Suprarenal capsule by mouth.	None	None	None	Only effect of treatment was to make a subnormal temperature normal.

No. of Case.	Reference.	How employed.	Effect on Pulse.	Effect on General Symptoms.	Effect on Pigmentation.	Ultimate Result.
13	L. A. Francis. *Brit. Med. Journ.*, May 2, 1896. (Male, 25.)	5-grn. suprarenal tablets by mouth. 3 *per diem* increased gradually to 6.	Improved at first	Improvement at first along with gain in weight (5½ lbs. in two weeks).	At first lessened, but fresh patches afterwards appeared.	Marked improvement for 3½ months. Then relapse and somewhat sudden death.
14	Ringer and Phear. *Clin. Soc. Trans.* Vol. XXIX., p. 68. (Female, 28.)	3 pills daily; each pill = 15-grn. suprarenal body. Ultimately she took equivalent of ¼ oz. suprarenal daily.	No rise of pulse-tension	Temporary improvement at first. But lost 13 lbs. even during this period of apparent betterment.	Much less marked, so that "the aspect would now hardly suggest Addison's disease."	After 4 weeks change for worse. Treatment began July 9. Death, Aug. 18. Diagnosis confirmed P.M.
15	Judson, S. Bury. *Lancet*, June 19, 1897. (Female, 13.)	½ tabloid suprarenal extract *t.d.s.* Afterwards a glycerine-extract of fresh gland used.	No improvement. Became weaker.	No improvement in general symptoms.	—	Treatment during 9 days. No benefit. Convulsions and death. Diagnosis confirmed P.M.
16	Henry Waldo. *Brit. Med. Journ.*, July 10, 1897. (Male, 36.)	Suprarenal tabloids by mouth. (Only given day before death.)	—	—	—	No result.
17	Ralph Stockman. *Edin. Med. Journ.*, 1867, Vol. I., p. 211.	Presumably by mouth	—	—	—	No improvement. Diagnosis confirmed P.M.
18	Osler. *Johns Hopkins Hosp. Bulletin*, Nov. to Dec., 1896, p. 208. (Female, 21.)	½ gland *per diem*. Glycerine-extract.	Pulse kept very weak throughout.	No benefit	—	Girl died suddenly on ninth day of treatment with delirium and collapse
19	Charlewood Turner. *Lancet*, June 10, 1899. (Male, 53.)	Glycerine-extract of pig's suprarenal.	None	Patient made very sick	—	Died in three days.
20	E. G. Trevithick. *Lancet*, July 14, 1900. (Male, 40.)	Suprarenal extract used twice a day late in disease.	None	None	None	Did not avert a fatal result. P.M.: Adrenals. Dense fibrous tissue with small centres of caseation.

21	Strümpell. *Text-book of Med.*, 3rd edition, 1901, p. 653.)	Adrenal tablets by mouth for a considerable period	No improvement -	None - - -	None - - -	No advantage from the treatment.
22	Anderodias. *Journ. de Méd. de Bordeaux*, Juillet 1, 1900. Obs. II.	20 centigrammes fresh gland in pilules daily.	Pulse stronger and more rapid at first. Then diminished in frequency and strength.	Seemed rather less torpid two days after commencement of treatment.	—	Treated over 12 days. For first 2 or 3 days slight improvement. Then rapid decline and death. Diagnosis confirmed P.M. (Patient in last degree of cachexia.) Capsules large, soft, with cavities.
23–28 (Six cases)	Box. *Practitioner*, 1901, I. 530.	Given by mouth and occasionally by rectum.	Pulse-tension not increased.	Asthenia not benefited -	—	Not slightest improvement.
29	Ditto - -	Tabloids of extract dissolved and injected. Gave rise to painful indurated swellings and "parchment" skin.	None - - -	None - - -	None - -	Death ultimately from epileptiform convulsions.
30–32 Three cases	Osler. *Principles & Pract. Med.* Art. Addison's Disease.	Suprarenal extract - -	None -	None - - - -	None - -	Not the slightest benefit accrued.
33	W. Murrell. *Lancet*, Feb. 1, 1896. (Male, 31.)	Tabloids by mouth, 1 *t. d. s.* each = 5 gr. of extract. Later 4 *in die.* Later fresh capsules of sheep in ʒ j doses.	Pulse weak throughout, and no rise in arterial tension.	Lost weight and strength. Nausea and vomiting made worse.	Not lessened, but if anything increased.	Gradually sank and died, having been under treatment 53 days. Diagnosis confirmed P.M.
34	Turney. *Lancet*, Jan. 18, 1896. (Report of Clin. Soc. Lond.) (Male, 30.)	Glycerine extract of adrenals = ½ gland *per diem.*	None - - -	Asthenia not bettered. Vomiting made worse.	—	Death. Diagnosis confirmed P.M. Both adrenals caseous.
35	Fränkel. *Ein Beitrag z. Ther. des Morb. Ad. mit Nebennierenpräparaten*, 1900, p. 16. (Male, 23.)	Suprarenal gland of pig. Treatment for 14 days. Total given = 39·9 grms.	Nothing definite -	Lost weight slightly - -	—	"The result of the suprarenal feeding in this case = 0." But patient quite well and vigorous 3¼ years after discharge from klinik.

No. of Case.	Reference.	How employed.	Effect on Pulse.	Effect on General Symptoms.	Effect on Pigmentation.	Ultimate Result.
36	Fränkel. *Ein Beitrag z. Ther. des Morb. Ad. mit Nebennierenpräparaten*, 1900, p. 18. (Male, 35.)	Tabloids of suprarenal substance (B. W. & Co.) 1-3 daily. By mouth. Under observation 7½ months.	No very definite effect	Felt much better. Appetite improved. Weight increased. Diarrhœa occasionally.	Colour lightened from that of a Nubian to that of a very sunburnt man.	Apparent temporary benefit, but Fränkel does not ascribe any specific action to the suprarenal preparation. Ultimate fate unknown.
37	Hamel. *Deut. Archiv. für Klin. Med.*, 1901, p. 241 (Vol. 71). (Male, 30.)	Tablets of compressed substance. 1 tablet 3 times a day "*per os und per clysma.*"	—	—	—	No perceptible result. Died.
38	Pickardt. *Berlin klin. Woch.* Aug. 15, 1898. (Male, 34.)	Tabloids (B. W. & Co.), containing ·3 grms. Only used over a few days.	—	Decrease of weight. Notable disturbance of nitrogenous equilibrium. (No signs in lungs.)	—	Negative. Patient died. No P.M., but diagnosis reasonably certain.
39	Althaus. *Med. Soc. Lond.*, Nov. 11, 1895.	Suprarenal extract	—	Improvement at first	—	Relapsed and died.
40	Dezirot *Thèse de Paris*, 1898, p. 57. (Case under care of MM. Variot et Peyré.) (Female, 15.)	Injections 2 c.c. suprarenal juice morning and evening, then once daily.	—	Improved somewhat	Slightly diminished	Sudden death in less than a month.
41	Longworth. *Brit. Med. Journ.*, July 19, 1902.	—	—	—	—	No benefit. Used late in disease. P.M.: Medulla of both suprarenals entirely fibrous.
42	Seibert. *New-Yorker Med. Monat.*, No. 3, 1899, p. 131.	5 grns. suprarenal extract 3 times a day.	—	—	Lessened	Not stated. (Diagnosis also apparently doubtful.)
43	Thomas Oliver. *Internat. Clinics*, Vol. II., 4th Series (1894).	Subcutaneous injections of suprarenal juice of rabbits. Afterwards 2 injections of adrenal extract from sheep. Under treatment 22 July to end September.	Not stated	No striking result. "No marked gain in strength and weight." Diuresis at first, and increase of urea excreted.	Pigmentation of face distinctly lessened.	Seen rather over 4 months after leaving infirmary. Pigmentation increased. Rising pulse. Emaciated.

44	Dupaigne. *Opothérapie Surrénale*, 1896, p. 109.	'2-3 grammes of capsule daily. (Had advanced tuberculosis of lungs.)	—	—	—	No benefit, but no inconvenience from the treatment.
45	Kindly communicated by Dr. D. N. W. Murray.	Suprarenal tabloids by mouth.	Presumably none	None. Vomiting persisted, accompanied by severe attacks of abdominal pain. Pain also in region of suprarenals.	None	Gradually sank and died. P.M.: "Well-marked caseating foci were found in both suprarenals." No sign of tubercle elsewhere.
46	Dyson. *Quart. Med. Journ.*, 1894. III., 43. (Female, 13.)	Tincture of suprarenal gland by mouth.	None	None	None	Patient in last stage of asthenia. Only took a few doses, which did not delay the fatal termination. P.M.: Suprarenals calcified, and 1 fibrous.
47	Dupaigne. *Op. cit.*, p. 102. (Oral communication of M. Faisans.)	Not clear	—	None	Disappeared	Only effect was the disappearance of the melanodermia.
48	Kinnicut *Am. Journ. Med. Sci.*, July, 1897.	Not stated	None	None	None	No effect, but treatment only initiated with development of toxic symptoms. Death 5 days later. P.M.: General tuberculosis, tubercular and fibroid changes in adrenals.
49	Communicated by Dr. John Hay. (Dr. Barr's patient.) (Male, 35.)	Suprarenal tabloids 1 *t. i. d.* Commenced March 22, 1897.	Somewhat better tension at first, but no permanent increase.	On May 5, discharged to convalescent home feeling decidedly better. Less faint. Returned with a relapse. Tabloids ineffectual.	"Pigmentation decidedly less," both on body and in mouth. Skin again become darker a few days before death.	Complicating R. apical phthisis. Died of asthenia Oct. 2. P.M.: Tubercular suprarenals. Tuberculosis both apices.
*50	Ditto	Suprarenal tabloids, 1 *t. i. d.* Commenced Dec 14. Left hospital Dec. 21.	No very definite effect	Pains relieved. Felt well and slept well.	—	Dec. 21. Left hospital at own request somewhat improved.

GROUP III.

Cases in which marked Improvement coincided with Organotherapy.

No. of Case.	Reference.	How employed.	Other Treatment used.	Effect on Pulse.	Effect on General Condition.	Effect on Pigmentation.	Ultimate Result.
*51	Lloyd Jones. *Brit. Med. Journ.*, Aug. 24, 1885. (Female)	Suprarenal tabloids (B. W. & Co.), 3 increased to 12. Later a 1 in 2 extract, 1½ oz. daily.	Iodide of mercury at first. Later the perchloride with iodide of sodium and reduced iron.	—	Vastly improved. Sickness stopped, appetite good.	Excessive pigmentation has quite disappeared.	"Appears to be practically well." (But *see* below.)
*52	Oliver. *Pulse Gauging*, 1895.	—	—	Increase in radial calibre.	Gain in weight. Disappearance of nausea and anorexia.	Lessened - -	Dr. Oliver informs me patient has been lost sight of.
*53	Rolleston. *Brit. Med. Journ.*, April 6, 1895. (Female.)	Suprarenal extract by mouth.	—	—	Stronger and less subject to nausea and vomiting.	Still pigmented -	Under treatment 10 months. On the whole decidedly better. (*See* next section.)
*54	Chas G. Stockton. *Med. News*, Sept. 16, 1895.	Two uncooked adrenals of sheep daily.	Oxygen, iron, arsenic.	Most striking improvement. Became large, strong and frequent. B.-P. raised beyond that of health.	Decided increase of strength and return of appetite.	Ultimately almost entirely disappeared.	Practically well. Under observation June to October. (*See* postscript.)
55	Byrom Bramwell. *Brit. Med. Journ.*, 1897, Vol. I., p. 68. (Male, 37.)	Hypodermic injection of extract - 1½ rabbits' suprarenal alternately over kidney-regions. Also juice by mouth.	"Levico" water (arsenio-ferrated).	Markedly improved	Marked improvement -	Greatly lessened -	Death from influenza (11 months after). P.M.: Absence of suprarenals; place taken by masses of fat.

56	Spillman. *Revue Méd. de l'Est*, Jan. 15, 1896. (Male, 44.)	Hypodermic injection of suprarenal juice in dorso-lumbar regions. (May 7th to July 4th.)	Tonic regimen of glycerophosphate of lime and an iodo-arsenic solution.	—	Markedly lessened asthenia. Rapid increase of weight 4 kilogs. *Retour des forces, de la gaité, de l'energie.*	Notable diminution -	Great amelioration. Ultimately relapse, death by syncope. No P.M.
57	Widal. Report in *Gaz. des Hôpitaux*, Mar. 1, 1898. (Soc. Med des Hôpitaux.)	Not stated, but presumably by mouth.	—	—	Asthenia bettered in a few days to such an extent that patient, who was at first unable to walk, was able to do so for some time at a stretch.	None - - -	Not given.
58	Edel. *München med. Woch.*, Dec. 25, 1900. (Male, 33.)	Tabloids of suprarenal substance 2 *bis die.*	None mentioned -	—	Able to resume heavy work. (Had been ill 6 months, got steadily worse during that time. Began to improve rapidly within 2 or 3 days of inception of treatment.)	Progressive clearing up from third day of treatment. About fifth week only around eyes and in mucous membranes. Bronzing returned in former intensity when the extract was stopped.	Died from tubercular meningitis. P.M.: Tubercular nodule size of pea in right suprarenal only.
*59	Anderodias. *Journ. de Méd. de Bordeaux*, Juillet 15, 1900. (Female, 66.) Observation V.	By mouth in pilules -	—	Pulse - tension slightly raised.	Weakness lessened -	Almost disappeared -	For some weeks no appreciable improvement. Ultimately general condition greatly improved and patient no longer kept her bed.
60	Anderodias. *Journ. de Méd de Bordeaux*, Juin 24, 1900. (Male, 26.) Observation I.	In pilules by mouth = 10 cgms. fresh gland daily. Commencing with 1 pill.	Rest and at first a tonic.	Good effects in 5 or 6 days. Tension increased. Cardiac bruits less marked.	In third month strength completely reëstablished. Pains disappeared. Appetite good.	Bronzing notably lessened.	In middle of fourth month felt perfectly well. Took up the fairly heavy trade of farrier.
61	Dezirot. *Thèse de Paris*, 1898. (Male, 14.) Patient of M. Variot, p. 52.	Injections 3 times a week. Later dry extract. Then again hypodermic injection.	Iron, quinine, and cod-liver-oil also given, Fowler's solution.	Pulse kept feeble throughout and extremities always cold.	Patient felt stronger, but no objective increase in strength. No pains, no gastro-intestinal troubles.	Notably diminished on backs of hands, face, and neck.	Sensible improvement during the 10 months of observation.

No. of Case.	Reference.	How employed.	Other Treatment used.	Effect on Pulse.	Effect on General Condition.	Effect on Pigmentation.	Ultimate Result.
62	Dezirot. Patient of MM. Variot and Comby, p. 55. (Male, 14.)	Daily ingestion of a suprarenal capsule of sheep.	None mentioned -	Pulse regular, 68 per minute.	Digestive troubles lessened, pains diminished and muscular strength augmented.	No especial effect -	Great amelioration. Patient lost sight of after some months.
*63	Sansom. *Med. Soc. Lond.*, Nov. 1895.	Tablets of suprarenal extract, 1–2 t.d.s.	Previous treatment with arsenic had been without benefit (lost 1½ lbs. in fortnight).	—	Undoubted general improvement, gained 14 lbs. within a month.	—	Not stated. (*See* ensuing section.)
64	Vollbracht. *Wiener klin. Woch.*, July 13, 1899. (Morb. Ad. following Purpura Hæmorrhagica.)	Suprarenal pastilles 3 daily, increased to 4. (Continued for 32 days.)	—	Pulse became normal.	Improvement for a time. Increase in weight.	Pigmented spots on mucous membrane of mouth distinctly paler.	29 July. Patient left hospital much better. 6 Sept. Relapse. 13 Sept. Died with symptoms of tubercular meningitis. Diagnosis confirmed P.M., but no meningitis. Suprarenals caseous, especially right.
65	Fränkel. *Op. cit.*, p. 22. (Basedow's disease with symptoms of Addison's.)	Suprarenal tablets by mouth.	Thyroid tablets also given.	Palpitation and syncopal attacks became less frequent.	After many vicissitudes the asthenia and gastro-intestinal symptoms markedly improved.	Pigmentation waned -	Marked betterment all round.
66	Kaufmann. *Centralblatt für Stoffwech. und Verd. Krank.*, No. 7, 1901, p. 173. (Male, 20.)	Merck's suprarenal tablets by mouth, 15 to 25 tablets. Treatment began Nov. 29, 1900.	Hygienic measures	—	Weight rose from 49 to 54 kilogs. Appetite became excellent. Asthenia removed. Patient went back to work and treatment discontinued Jan. 28, 1901.	Pigmentation distinctly lessened.	Died with toxæmic symptoms and heart failure, March 4, 1901. P.M.: Suprarenals completely caseous. All other organs sound.

67	Schilling. *Münch. med. Woch.*, Feb. 16, 1897.	Suprarenals of a young animal (lamb) minced fine and given raw with some beef. 1 *per diem*. Begun Jan. 1, 1896, with ½ gland.	Arsenic had been tried with but very temporary benefit.	Heart's tone improved.	Asthenia disappeared. Appetite regained. Increase of over 30 lbs. in body weight.	Skin returned almost to its normal colour.	Patient went to work against advice. Soon after became suddenly ill with toxic symptoms and heart-failure and died June 27, 1896. P.M.: Suprarenals completely shrivelled up and replaced by scar-tissue, which was permeated by dry cheesy material.
68	Senator. *Charité Annalen*, 1897. (Quoted by Frankel *op. cit.*)	Tablets of suprarenal substance of wether = 1·2 to 2·7 grms. of fresh gland daily.	—	—	Asthenia almost completely disappeared. Weight rose 5 kilogs. Appetite much bettered.	None - - -	Under treatment 18 days. Greatly improved. (Whether due to the opotherapy doubtful.)
69	Dieulafoy. *Cours de Pathologie Interne.* Tome III., p. 289 (1901). (Communicated by Pech.)	"Capsules surrénales de bœuf preparés en brochette et rôtis sur le gril."	—	—	Surprising amelioration. Patient from being in an advanced state of asthenia was able to go upstairs and to walk without fatigue. Appetite extremely good.	Skin lost its bronze coloration.	Very greatly benefited in all respects.
70	Ditto. (Dieulafoy's own case.) Tome III., p. 287. (Male, 29.)	1 c.c. suprarenal extract every other day. Also gelatine pilules containing ·1 grm. desiccated gland 4 *per diem*.	Cod-liver oil also used. (Patient also had pulmonary tuberculosis.)	—	Rapid improvement. Asthenia bettered little-by little. Weight increased from 48 to 61 kilogs. Two relapses on resuming work quickly relieved each time by continuation of treatment.	Pigmentation not removed.	Indefinite. Improved markedly each time under treatment.
71	Dr. H. D. Rolleston (kindly communicated.) (Male, 20.)	Suprarenal extract -	—	(Radial pulse absent for 24 hours before death.)	Improvement very marked for 2 months.	—	Sudden collapse and death.

No. of Case.	Reference.	How employed.	Other Treatment used.	Effect on Pulse.	Effect on General Condition.	Effect on Pigmentation.	Ultimate Result.
72	Dr. H. D. Rolleston (communicated). (Male, 31.)	Suprarenal extract	—	(Radial pulse absent for 36 hours before death.)	Improvement at first. When suprarenal prep. pushed muscles became "rather stiff as if the physiological effect of contracture was produced."	—	Ultimately sudden collapse and death.
73	Epelbaum. *Thèse de Paris*, 1895. (Case of P. Marie.) (Female, 32.)	Fresh suprarenal capsules in bouillon or with potatoes or bread. Commenced June 21.	No other treatment mentioned.	Pulse-rate accelerated from 76 to 92 (average 86).	Almost immediate improvement in strength followed by relapse. Ultimately weakness, fatigue, and pains completely disappeared. Gain in weight.	None	General state very good. August 7.
74	Ditto. (Male, 26)	Suprarenal capsules by mouth. Begun June 10.	No other treatment mentioned.	Some rise in tension. Varying frequency.	Manifest gain in strength commencing after 10 days. Gain in weight. Diuresis.	Pigmentation stationary.	Amelioration manifest. Able to return to work. July 19.
75	Dupaigne. *Opothérapie surrénale Thèse*, 1896. (Paris.) (Female 35.)	Fresh glands of calf. Later glands "finement hachées." Begun Jan. 16. Dose, ·8 to 3 grms.	Occasional inhalations of oxygen.	Pulse-tension rose from 12 to 18 (sphygmometer.)	Very much improved. Mar. 27. Fatigue-curve quite normal (by ergograph). Weight augmented. Appetite continually good and no gastric troubles.	General tint lightened	Went out April 14. She omitted treatment. Died April 22 with toxic symptoms. No P.M.
*76	Box. *Practitioner, loc. cit.*	By mouth and hypodermic injection.	—	—	Some improvement as regards asthenia and general condition.	Some improvement	Dr. Box states that the ultimate fate of patient is not known.

77	Ebstein. *Deut. Med. Woch.* Nov. 11, 1897.	Daily administration of 3 suprarenal tablets (Merck's).	—	—	Able to do housework again and for a time felt quite well.	Pigmentation of skin and mucous membranes lessened.	Case ultimately fatal.
78	Mahé. *Thèse de Paris*, 1894, p. 48. (Communicated by M. Langlois.) (Male, 37.)	Injections every other day of 1 to 3 cc. suprarenal liquid.	Rest and oxygen inhalations.	—	Strength undoubtedly increased. Appetite returned.	Not altered - -	Amelioration vanished a few days after leaving hospital. Ultimate fate unknown.
79	Anderodias. "*Quelques Results de l'opoth. surrénale dans le trait. de la mal. d'Addis.*" 1900. Observation III. (Female, 64.)	Pilules suprarenal extract = 10 cgr., fresh gland daily. Increased to 20 cgr. (Begun March.)	Milk-diet and rest to combat digestive and renal trouble.	—	Strength became almost normal. Relapse on discontinuing extract. Strength again reëstablished on resuming treatment.	At third month skin almost normal except on back, where patient lay.	From being in an advanced stage of asthenia became well, and continued so when last seen (Dec.) in spite of heavy work. Now lost sight of. Continued the extract daily when last heard of.
80	Anderodias, *op. cit.* Observation IV. (Male, 38.)	Capsules of the extract. Commenced 8 June.	Apparently none -	Pulse-frequency increased from 52 to 72.	Strength returned. Able to return to work. Has not kept his bed a single day since leaving hospital. Pains disappeared.	Slightly diminished -	Well when seen in October. Still taking 2 capsules of extract daily. (Lungs were normal.)
81	Shoemaker, *Journ. Am. Med. Assoc.*, March 23, 1901. (Male, 19.)	Tablets suprarenal extract, 5 grms. Two per day, and dose gradually increased.	None. Not even rest in bed.	Not stated -	Improved markedly for 3½ months. Strength somewhat increased. Gained 6 lbs. in weight. Nausea and vomiting abolished.	Discoloration of skin less decided.	Relapsed, and died on third day of relapse. P.M.: Adrenals partly caseous, partly fibroid. Pulmonary tuberculosis.

GROUP IV.

Cases in which permanent Benefit accrued apparently as a Result of the Treatment (? Cures).

No. of Case.	Reference.	How employed.	Other Treatment used.	Effect on Pulse.	Effect on General Condition.	Effect on Pigmentation.	Ultimate Result.
*82	Oliver. "*Pulse Gauging*," 1895. (Seen with Mackern.)	Suprarenal pills = gr. xv. suprarenal medulla. 2 *per diem.*	—	Increase in radial calibre.	Gain in weight. Disappearance of nausea and anorexia.	Lessened - -	Permanently benefited. (*See* "Additional information.")
*83	Ralph Stockman. *Edin. Med. Journ.*, Vol. I., 1897, p. 211.	First 3, then 6, raw sheep capsules given daily by mouth.	—	—	Greatly improved within 2 months.	Markedly lessened -	Improvement maintained for 20 months. Relapses occurred when extract omitted. Case due to injury. (Dr. Stockman informs me case has now been lost sight of.)
*84	Suckling. *Brit. Med. Journ.*, May 28, 1898.	Tabloids suprarenal substance, gr. v. *bis die.* Begun April 1897. Gradually increased to 20 to 35 tablets a day.	—	Pulse became normal.	Very great improvement. Asthenia disappeared.	Pigmentation of skin almost gone.	Permanent improvement. (*See* under "Additional information.")
85	Sellier and Vergier. *Journ. de Méd. de Bordeaux*, October 23, 1898. (Male, 31.)	Begun June 1897. By mouth Glycerine extract and dry extract used = 2 grms.; fresh gland daily. Treatment finally discontinued August 15, 1898.	Treated by high altitudes for the accompanying pulmonary tuberculosis.	Arterial pressure not measured. No modification of cardiac rhythm.	Strength completely restored. Lumbar pains rapidly ameliorated. Appetite returned. Gain in weight.	Tint lightened early in treatment. Macules in lumbar region disappeared. Elsewhere bronzing lessened.	August 15, 1898. "No pains, no digestive troubles; strength completely restored."
*86	Anders. *Text-book of Pract. of Med.*, 4th edition; and information kindly sent by Professor Anders.	By mouth. Suprarenal extract.	None mentioned -	Improvement -	Great improvement. Restored to "almost his usual vigour, both in mind and body."	Pigmentation not removed.	Not seen for a year, but under observation 2 years, and well during that time.

87	W. H. Thomson. *Med. News*, Vol. LXXVII., p. 357. (*New York Acad. Med.*)	—	—	—	Marked improvement -	Not stated - -	Case came under treatment 4 years ago. Has continued in reasonably good health up to date of paper (1900). Diagnosis not doubtful.
88	W. H. Thomson. *Med. News*, Vol. LXXVII., p. 357. (*New York Acad. Med.*)	—	—	—	"Intense tiredness and disinclination for exertion." Very markedly relieved.	—	Now able to resume employment (theatrical manager). Improvement has been maintained 3 years (1900). Diagnosis reasonably certain.
89	Hayem. *Séance de l'Acad. de Méd.* Reported in *Gaz. des Hôpitaux*, March 1, 1898.	½ oz. suprarenal capsule of cow daily. Also injections of a watery glycerine extract.	—	—	Strength returned to a great extent.	Bronzing unchanged -	Treated in 1894. Improved up to date of report, 1898.
90	Fr. Robin. *Thèse de Paris*, 1898.	Suprarenal capsules of sheep, calf, and ox. By mouth. Also intramuscular and hypodermic injections of glycerine extract. Under treatment nearly 5 months.	Rest -	No details given	Strength returned. Gastro - intestinal symptoms sometimes aggravated. Ultimately appetite improved.	Diminished. Pigmented plaques vanished.	"Cured." Remained well during 3 years of observation.
91	Anderodias. *L'Opothérapie surrénale dans le mal. Addison*, 1900. Observation VI.	Pilules = 10 cgms. fresh gland, one daily. Then two	No other treatment mentioned.	Pulse became stronger.	Asthenia completely disappeared. Digestive trouble entirely gone.	Tint perceptibly lightened on thorax and limbs.	Left the hospital completely reëstablished (October 1899). (*See* also under "Additional information.")
92	Seibert. *New Yorker med. Monat.*, No. 3, 1899. (*Deut. Med. Gesellsch. von N. York.*) (Male, 40.)	Suprarenal extract, 5 grms. *t. d. s.*	Treated exclusively with suprarenal for a year.	—	Gastro-intestinal symptoms only very seldom. Gained 20 lbs. Hand-grips distinctly stronger.	Colour has become distinctly clearer; the dark spots in mucous membrane of mouth have almost disappeared.	Very great improvement. Has only lost 2 weeks during the 12 months of treatment.

No. of Case.	Reference.	How employed.	Other Treatment used.	Effect on Pulse.	Effect on General Condition.	Effect on Pigmentation.	Ultimate Result.
*93	*Report*, 1901, *The Infirmary, Leicester.* (Female, 17.)	Suprarenal tabloids by mouth; 1 to 4 three times a day.	None. (No lung-affection.)	No special effect -	Gradually became stronger. Sickness stopped. Pain abolished.	Not diminished - -	Steady improvement. Twice under treatment. Improved greatly each time. (See also "Additional information.")
*94	Dr. William Osler (kindly communicated).	Suprarenal extract by mouth. (July, 1899, to November, 1901).	Rest in bed at first	Frequency lessened. (120 to 90.)	Little change at first. Ultimately marked and continued improvement, and patient went back to his work.	"No increase in pigmentation, which, if anything, was less."	Death from an acute infection (probably Friedlander's organism), November 1901. P.M. "Adrenals sclerotic, and had almost completely disappeared." (*See* also "Additional information.") Lungs normal.
95	Osler. *Internat. Med. Mag.*, February 1896. Case VI. Also *Prin. and Pract. Med.*" Article: Addison's Disease.	Treated for 8 months with glycerine extract of pig's suprarenal. By mouth. ½ to 3 glands, *t. d. s.*	Rest - -	Pulse from 120 to 130, brought down to 84 to 104.	Great improvement. "Was bright and active, and said he felt vigorous." Active and energetic. Gained 19 lbs. during treatment.	Not removed, but some small patches on palate disappeared, and colour less intense on trunk.	Improvement maintained. Was alive 2 years subsequently. (Pulmonary tuberculosis complicated.)
96	M. E. Boinet. *Soc. de Biologie*, November 11, 1899. (Male, 35.)	1 cc. capsular liquid. Perf. after method of Brown-Séquard, and injected. Begun April 1, 1898.	Also a course of hydrotherapy.	—	General state became good. Asthenia almost disappeared. Long walks and excursions became possible.	Lessened - -	November, 1899. Very great improvement. Tremors of hand and fingers induced when dose increased; still remain, though less.
97	Bate. *American Pract. and News*, August 1, 1899.	½ grn. suprarenal extract. *t. d. s.* Treatment for 2 years at intervals.	—	Faintness relieved.	Asthenia and nausea almost disappeared. Relapsed twice when extract could not be obtained.	Greatly lessened - :	Very great and lasting improvement.

ADDITIONAL INFORMATION RESPECTING CASES IN THE ABOVE LIST.

Case 51.—Dr. Lloyd Jones writes: "The case to which you refer passed out of my observation for some months, during which time she resumed the rather arduous duties of a bedmaker. When I next saw her, I diagnosed the case as one of pernicious anæmia, and of pernicious anæmia she died, under the care of one of my colleagues at the hospital. I would much like to know what the primary ailment was. When I first saw her she was much pigmented, fainting often, sick, with a thready rapid pulse; and all these signs disappeared for good while she took one or two ounces of a 1-in-2 extract of suprarenal daily." I am not informed that a *post-mortem* was obtained. The difficulty in distinguishing pernicious anæmia from Addison's Disease has not seldom been found to be a very real one. Dr. Hale White,[1] speaking at the Clinical Society, London, January 10, 1896, mentions a case which he treated with suprarenal extract under the impression that it was one of Addison's Disease, but which proved eventually to be pernicious anæmia. In my own limited experience I have come across a similar case.

Case 53.—Dr. H. D. Rolleston writes: "The case referred to . . . was seen by me for some considerable time after that date (April 6, 1895), I think for two years, and then lost sight of; as far as I know, the case has not ended fatally. Of course the question arises whether after all it was a case of Addison's Disease, though if it had got worse there would have been no question." Dr. Rolleston has seen a number of cases since the one mentioned (*vide* cases 71 and 72) and has the impression that those in which the diagnosis is quite clear "often improve for a time under suprarenal extract, sometimes to an extraordinary degree, but that they subsequently relapse and die."

Cases 59 *and* 91.—Prof. Anderodias having made enquiries for me concerning the after-histories of his cases, has been able to learn some recent details in these two patients. Case 91 has been well since he left the hospital. Moreover, he carries on the rather heavy work of a "manœuvre" without fatigue. The diagnosis was not doubtful. The other patient is not so satisfactory. She is alternately better and worse, and is obliged from time to time to take a little of the suprarenal extract. But the improvement is yet striking enough.

Case 50, communicated by Dr. Hay, though improved under suprarenal medication, has been classed with Group II. because of the short period during which it was under observation (a week). He was admitted complaining of general weakness. The pulmonary signs were normal "except for slight dulness at right apex posteriorly," and there was slight albuminuria. He seemed to improve during the week when he was treated with suprarenal tabloids, but it is to be noted that while his temperature was 97° F. on admission, the maximum and minimum during his stay were 101·8° and 99° respectively, while he went out with the thermometer at 101° F.

Case 63.—Dr. Sansom informs me that in so far as he recollects, his patient was "*not persistently relieved* of the symptoms of Addison's Disease." He has not been very favourably impressed by the treatment with suprarenal glands, though the improvement was at first very great in his reported case. Dr. G. Wilson has been good enough to search the case-books of the London Hospital, but no further details are to be learnt regarding the case.

Case 82.—Dr. George Oliver has made enquiries from Dr. Mackern with reference to the subsequent progress of this patient, the result of which would seem to be unique. The patient has, it seems, been taking suprarenal tabloids for seven years, and (May, 1902) it is now about eight years since Dr. Oliver first prescribed them for her. "When the treatment was commenced," he writes, "the patient looked in a very bad way, and I did not think she could have survived more than a few months." Dr. Mackern reports that the woman is still "very much alive." She considers herself "in a much better state of health than some people who consider themselves very robust." . . . She has gone on steadily every day taking two of the suprarenal pills . . . (= gr. xv. suprarenal medulla). . . . Her skin is still dark, though the patches are not nearly so marked or the edges so defined. . . . She busies herself perpetually about the house"—and is able to undertake railway journeys frequently.

Case 84.—Dr. C. W. Suckling writes: "I am glad to say my patient is quite well. I saw him two or three months ago. (Date of letter is January 9, 1903.) He is in business and, as I say, well and able to walk perfectly well."

Case 86.—Prof. James Anders of Philadelphia has replied: "The preparations of suprarenal capsule exercised an excellent effect, restoring the man to almost his usual vigour, both in mind and body. I cannot state whether the man still maintains the improvement reported, inasmuch as I have not seen him for more than a year, although it must have continued for a period of two years before he dropped from observation."

Case 93.—Dr. D. N. W. Murray, house-physician at the Infirmary, Leicester, states: "The patient is still alive, and except for occasional attacks of vomiting is perfectly well and has improved greatly in health."

Case 45, also communicated by Dr. Murray, is interesting from the point of view of a simple, early, uncomplicated case of Addison's Disease which did not benefit in the slightest degree by organotherapy. He had been quite well until three months before he applied for treatment, and the physical examination gave and the *post-mortem* proved no lesion tubercular or other elsewhere.

Case 94.—The history of this case has been very kindly written out for me by Professor Osler, and I give it here in full owing to its intrinsic importance. I do so the more readily as the case has not been before published.

"On the morning of September 30, 1899, I saw, with Dr. Reinhart, J. E., aged 21, a commercial traveller.

"Family history good.

"The trouble began in April with pain in the left side and digestive disturbance. In May and June he had a great deal of nausea and vomiting, and on several occasions vertigo in the street.

"When Dr. Reinhart first saw him his pulse was extremely feeble, irregular, and intermittent. He had been in bed for between three and four months, the chief symptoms being gastric, with extreme irregularity and feebleness of the pulse. About three months ago (?) it was noticed that his colour was changing, particularly in the face.

"I found a delicate-looking young man, with marked pigmentation of the face, a line of pigmentation on the mucocutaneous surfaces of the lips (slight), and pigmentation inside the mucous membranes of both lips; none elsewhere; general surface not pigmented. The pulse was 120; an occasional intermission. Slight tenderness on deep pressure over the suprarenals. No sign of trouble in the lungs; he had slight fever during July and August. He had been taking suprarenal extract for a month or more, and had recently been much better. He began the extract in July, 1899. Little change until autumn of 1900. Off and on gastric symptoms and weakness and inability to do anything continued until early in 1901. He then began to get better and went to work. No increase in pigmentation, which, if anything, was less. Dr. Thayer saw him in October, 1901, looking practically well, except that the pigmentation still persisted; pulse under 90. In November he had an acute infection, sudden fever and delirium, and a general septicæmia, possibly with Friedländer's organism, and died in three or four days. He took the adrenal extract up to the time of his death. The adrenals were sclerotic and had completely disappeared." (For Case 54, *see* Postscript.)

REMARKS AND ANALYSIS.

Group I.—The experience with suprarenal grafts is seen to be untoward. There appear to be reasonable grounds for supposing that in these three cases death was consequent upon the operation. Jaboulay (*loc. cit.*) indeed expressly states that this was so in the two patients he saw. Examples of sudden death during the course of organotherapy (as case 7) are furnished also by cases 13, 18, 40, 71, and 72; but it must be here remembered that sudden death is a well-recognised mode of termination of Addison's Disease whether treated or untreated. J. Ihler[3] summarises a number of such cases, though he includes wrongly the 6th case of Addison. "Le patient mourut subitement" does not give the sense of Addison's own words: "The patient speedily sank."[4] Dieulafoy (*op. cit.*) also lays stress upon the occasional sudden termination of the disease, and makes the interesting statement that this is more likely to occur in the "*forme fruste*" of the disease, which term he applies when the bronzing of the skin is wanting. He quotes some illustrative cases. Anderson[5] and Bradbury[6] also record cases suddenly fatal where pigmentation was absent. In the first, the patient, a boy of 14, died suddenly from an attack of cardiac syncope. The post-mortem examination confirmed the diagnosis. In Dr. Bradbury's case death occurred suddenly while the man was being assisted into bed. No suprarenal medication had been used. In 3 out of 12 cases of Morbus Addisonii coming under the notice of Osler death was sudden. Severe cardiac distress, however, may be directly consequent upon the use of suprarenal preparations, and has been noted in cases other than this affection when thus treated.[7] On the whole, there is no evidence to show that any serious risk is incurred in the treatment of Addison's Disease by the ordinary methods of opotherapy.

Group II.—This is a large and dispiriting succession of failures. It probably represents the usual experience of organotherapy in Addison's Disease. We ought, however, on the one hand to "respect the burden" under which the method laboured in cases 8, 10, 16, 19, 20, 22, 41, 46, and 48, where it was tried either with the patient practically moribund or over but a very brief period of time, and on the other hand to remember that the number here collected ought probably to be multipled by no mean figure in order to arrive at an approximate notion as to the relative frequency of such results. For it is just this class of case which would ordinarily not be thought worth the recording. This would scarcely apply to the apparent successes.

Groups III. and IV.—Before attaching weight to the more or less successful results here summarised, it is necessary to recall certain well-known features of the disease itself. In the first place cases of apparent recovery have been observed long before the advent of "the method of Brown-Séquard." A striking instance is afforded by Dr. Finney's "Case of apparent recovery from Morbus Addisonii."[8] This is a very carefully-reported case, the diagnosis of which appears not in the least doubtful, in which the asthenia was bettered so far that the patient was able to walk two miles, and in which the pigmentary changes disappeared. The treatment consisted of a nourishing diet, with wine, &c., and the exhibition of iron and arsenic. Dr. Finney also refers to a similar case of McCall Anderson's.*

Nothing, moreover, is more remarkable in the natural history of the affection than the very complete remissions which are not very seldom seen. Greenhow,[9] speaking from an almost unique experience, insists upon the *paroxysmal mode of progress* with "alternate remissions and exacerbations." "During the remissions, strength is in a great degree recovered, the appetite improves, the sickness abates, the discoloration becomes paler; and, above all, the patient's whole aspect bespeaks that a heavy weight has been lifted from his head." This has recently been brought home to me in a disappointing manner. A promising case which a medical friend offered me to make observations upon happened at the time to be in a state of remission. At any rate her condition is at present so comparatively good as to render worthless any attempt to investigate the action of suprarenal preparations in her case. The markedly beneficial action of mere rest in bed has also been often noted.† It is obvious, therefore, that great caution is needed in interpreting the results in, at any rate, Group III. Only very extended observations are of any real value.

Even where observation has been reasonably extended, it is still necessary to be very cautious in interpreting appearances. Greenhow (*op. cit.*), writing before organotherapy was even thought of, remarks that the disease though "obviously incapable of cure . . . is, at the same time, *in no small degree amenable to treatment in respect of delaying its progress* . . . and it is impossible to say to what extent life may be prolonged under favourable circumstances."

In our list, Fränkel's case (No. 35) furnishes a good example of the fallacies which may surround an observation. Here the result of the suprarenal feeding was *nil*, yet on inquiry three and a quarter years after the patient's discharge from the *klinik*, he was reported quite well and vigorous. Had this improvement begun while under treatment, the case could scarcely have escaped being regarded as an instance of permanent improvement from organotherapy.

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* Dr. Rolleston, in Clifford Allbutt's *System of Medicine*, Vol. IV., p. 561, quotes Lewin's collection of 800 cases, among which 5 are reported as "cured" and 28 as improved.

† One of Chauffard's cases (*op. cit.*) on two occasions improved so markedly under mere rest alone that he felt able to return to work

An analysis of our material as it stands gives :—

Cases made distinctly worse - -	7
Cases deriving no real benefit - -	43
Cases showing marked improvement -	31
Cases permanently relieved (Group IV.) -	16

The two following tables include, of course, only those cases in our list where definite information is given as to the points at issue.

A.—TABLE showing coïncident progress of cases during the several methods by which treatment was carried out :—

Preparation and Mode of Administration.	No. of Cases.	Made Worse.	No Benefit.	Marked Improvement.	Permanent Benefit.
Suprarenal grafts - -	3	3 (fatal)	—	—	—
Fresh gland by mouth -	9	1	1	6	1
Hypodermic or intramuscular injections.	11	1	6	3	1
Fluid or solid extract by mouth.	61	2	32	17	10
Mixed methods (hypodermically and by mouth).	5	—	—	3	2
Unclassed - - -	8	—	—	—	—
Total - -	97				

B.—TABLE showing coïncident progress of cardinal symptoms during the treatment :—

Symptom.	Made Worse.	No Definite Alteration.	Improved.
Circulatory feebleness - -	2	21	20
Asthenia, &c. - - - - -	7	19	54
Pigmentation - - - -	1 (doubtful)	24	36

The pigmentation waned in most of the cases which improved, sometimes almost, if not quite, disappearing.

In only 6 cases out of the 20 reported as improved so far as regards the circulatory feebleness, it is definitely stated that the pulse-tension was raised.

Excluding those cases where organotherapy was instituted only in the last two or three days of illness there remain only 17 records of autopsies on patients in our list where more or less definite details as to the state of the adrenals were noted.

C.—TABLE showing *post-mortem* appearances of the adrenals in relation with previous reaction of the disease to organotherapy:—

State of Suprarenal Capsules.	No. of Cases.	Previous Reaction to Treatment.
(A.) Completely absent - -	2	Very markedly benefited (Both).
(B.) Shrunken and entirely fibrous.	2	One had benefited greatly; the other had derived distinct temporary benefit.
(C.) Tubercular nodule in one capsule.	1	Great amelioration.
(D.) Caseating foci and sclerotic changes.	5	Nil in 3. Temporary benefit in 2
(E.) Enlarged and caseous, with little or no fibroid change.	5	Nil in 3. Temporary benefit in 2.
(F.) Simply stated as both "Tubercular."	2	Nil.

In (*a*) and (*b*) there appeared to be no tubercular lesions elsewhere. The case (*c*) was carried off by tubercular Meningitis; the bronchial, retroperitoneal and other lymph-glands were caseous. In (*d*) microscopical examination proved that there was practically no normal medulla at all in two of the three cases where benefit was *nil*; in the other two the alteration was mainly fibrous, approximating to those included under (*b*). In (*e*) four of the cases are stated to have had either no tubercle elsewhere or no pulmonary affection. Two improved a little, two did not. In (*f*) there was tubercle in the lungs in both cases.

The various animals used to supply the glands used medicinally have been the dog, rabbit, calf, pig, lamb, ox, and cow, and in many instances it is specified that the medulla of the gland was used. In not a few cases, too, it is expressly stated that the suprarenals were from young animals.

In four instances interesting accounts have been given of the effect of the suprarenal feeding upon the nitrogenous metabolism. Pickardt (*loc. cit.*) states that in his case, immediately upon the institution of the "specific" treatment an important loss of the body-proteïd began, and the nitrogenous equilibrium which had been obtained in the "*vorperiod*" was seriously disturbed. The defect reached in 4 days 18·02 grms. N. equivalent to 120 grms. albumen. The nitrogen-output in the urine progressively increased from the first to the fourth day. The experiences however of Vollbracht, Kaufmann, and Senator (*vide* references) have been much more favourable. In Vollbracht's patient during a period of 7 days there was an average + N. of 5·72 grms (= 36 grms. albumen on the day's account), and the body-weight rose from 49·6 kilogs. on the first day to 51 kilogs. on the last. Kaufmann divided up his research into five periods. In a "*vorperiode*" of 9 days the nitrogen-balance per day was – 0·42 grms. In the first "*rhachitol*" period of 3 days (15 tablets *per diem*) the N.-balance was – 1·23 grms. In a second "*rhachitol*" period of 5 days (20 tablets) the N.-balance was + 0·42 grms. per day. In a third "*rhachitol*" period of 3 days (25 tablets) the N.-balance was + 0·51 grms. per day. Treatment was then discontinued, and in a "*nachperiode*" of 5 days the N.-balance worked out to + ·74 grms. per day. Senator's results also agree in the main with those of these two observers.

SUMMARY AND CONCLUSIONS.

We may first state a few considerations which must be balanced against those enumerated in the first part of the preceding section. Although cases of permanent improvement or even of apparent recovery have now and again been recorded before the advent of organotherapy, yet these are extremely rare. Dr. Finney (*op. cit.*), writing in 1882, admits that it is with the greatest difficulty that he has collected one single case other than his own. Shoemaker (*loc. cit.*) in his paper, however, alludes to another and more recent example, and one or two recoveries have lately been reported as the result of surgical interventions. Now, however, reports of more or less permanent recovery under "specific" treatment are, comparatively speaking, not so very rare, as our list testifies. It is reasonable to suppose that examples of marked improvement in such a disease would be no more likely to be left unrecorded in the pre-organic period than at the present time. One must of course discount the fact that in the case of a new treatment, results *will* happen in spite of the most elaborate precautions.

The next point is, how are we to distinguish in this list between "remissions" coïncident with treatment and improvements consequent upon it? We can but trust that the sum of a series of similar coïncidences is more than a coïncidence. It would be strange that all or even the majority of the relations between treatment and improvement recorded in Groups III. and IV. should prove to be mere coïncidences. We may, too, remember the fact long ago pointed out by Greenhow that "remissions" in a case of Addison's Disease are in general but preludes to lower levels, like the ups and downs of a switchback. It was very different with some of our cases.

There is also the disturbing factor of "other treatment used" to allow for. The "other treatment" in many cases, where this is given, was rest and general hygienic measures. It is almost impossible to exaggerate the importance of these measures, but they must necessarily complicate the deductions drawn from the employment of any drug whatever, whether in Addison's or a great number of other diseases. We are neither more nor are we less embarrassed in the particular case we are considering than in many another therapeutic problem.

Perhaps the most vital question, however, is: Were the examples collected in Groups III. and IV. cases of Addison's Disease at all? Who can tell without examining their suprarenals? This disease is not like Myxœdema, where a necropsy is scarcely necessary to complete the diagnosis. One of the cases, that of Dr. Lloyd Jones, has on further enquiry already broken down, and I must again express my gratitude for the valuable and instructive letter in which Dr. Jones answered my queries. Dr. Rolleston again remarks of his own case: "Of course, the question arises whether after all it was a case of Addison's Disease, though if it had got worse there would have been no question." Perhaps additional and conclusive evidence one way or the other may eventually be furnished by the post-mortem reports in the cases yet living. Till then, while admitting that diagnostic accidents are occasionally inevitable, we have no sufficient justification for maintaining a resolutely sceptical attitude towards these groups as a whole.

Moreover, there are already some instructive records of autopsies available. More especially fortunate is it that in two cases of our collection where the evidence of benefit was least open to objection, autopsies made owing to the deaths of the patients from other diseases have confirmed the diagnosis of Addison's Disease. I refer to Osler's and Byrom Bramwell's patients (cases 55 and 94). In Dr. Osler's account the only doubt which rises in one's mind is whether the "acute infection" which carried off the patient may not possibly have been the acute toxæmia which sometimes has supervened without apparent warning, and made an end of the victims of this disease. But I have no right to say this. First-hand impressions on the spot must necessarily outweigh second-hand ones at a distance. It is curious that a sudden attack of "influenza" terminated also Dr. Bramwell's case. Less conclusive, though similar, evidence in favour of organotherapy is afforded also by cases 58, 64, 66, 67, and 81. (*Sectio cadaveris* in all.)

In spite, too, of the numerous failures, there would seem to be a very strong presumption that the treatment is not inert in suitable cases as furnished by the records of Nos. 59, 70, 80, 82, 83, and 97. In these patients the suprarenal preparation has been used over long periods of time, and it is found that it is still necessary to use it on pain of a relapse. Dieulafoy's case (70) relapsed twice when the adrenal substance was withdrawn, and as promptly improved when this was resumed. Anderodias' patient (80), when last heard of, remained well in spite of heavy work, but found it necessary to take daily some of the extract. Another of this author's cases (59) "is alternately better and worse, and is obliged from time to time to take a little of the suprarenal extract." In Stockman's case (83) relapses occurred when the extract was omitted. Oliver's patient (82) has been using adrenal pills for over eight years. Twice in Bate's case (97) the man became much worse owing to intermission of the treatment due to difficulty in obtaining the drug. The case for organotherapy, to my mind, has a surer foundation built upon evidence such as this, than when based upon improvement dated from a single course of treatment. One has always the fear of Fränkel's extraordinary case before one's eyes (No. 35).

Lastly, the subsequent good reports over long periods obtained upon our enquiry in cases 51, 53, 59, 82, 84, 86, 91, and 93 are, at least, encouraging.

Putting together the whole of the evidence here brought forward, whether for or against, and allowing it gently and slowly to simmer down, there yet remains a residual conviction that suprarenal medication has been of real value in a certain class of cases of Addison's Disease. In what class? To this important question but an imperfect and unsatisfactory answer can as yet be given. It has been attempted by a careful comparison of the histories of cases in which opotherapy has failed with those in which it has seemed to do real good to find some indications which would help towards a solution. But the endeavour has not been very successful, and I am without the invaluable aid which a large first-hand experience can alone give. The treatment has apparently been signally successful in some of the least promising cases, where it has been used late in the disease and upon patients who have had complicating tubercular lesions elsewhere. Even an extreme degree of asthenia has not always militated against a degree of success which has seemed quite extraordinary, as evidenced by case 59. On the other hand, it has as signally failed in the simple uncomplicated disease taken quite early. These contradictions can scarcely as yet be reconciled. In four cases where it is definitely stated that the affection followed upon injury or over-exertion, these all improved markedly, though caution is required in reasoning from so small a number.

If we look to the preparation used we shall find this most diverse, and its modes of administration various. Most of the cases in Group IV., however, were solely treated by the administration of the substance by the mouth, and its hypodermic use is seen to be weighted by no light objections. Moreover, recent research has appeared to show that the active principle is oxidised and rendered inert when injected into the tissues. The newly discovered active principles, Epinephrin (Abel)[10] or Adrenalin (Takamine),[11] would not appear to have been at present tried. Turning to the post-mortem records, where these are available, Table C. would seem to suggest that the cases most likely to derive benefit are those in which the tubercular process is a chronic sclerosing one, and where the other organs are fairly sound. This, in the main, embraces both Byrom Bramwell's and Shoemaker's points, though in view of the material benefit derived by some cases, in two of which the suprarenals have been found absent, in others reduced to fibrous tissue, we cannot in its entirety accept the latter's supposition that cases "in which organotherapy is useful are probably those . . . in which a portion of the gland or glands are (*sic*) functionally potent" (*loc. cit.*).

SUMMARY.

It would be wrong to call the following statements "Conclusions." They are merely deductions from the evidence before us:—

(1.) There would appear to be a certain class of case of Addison's Disease which derives indubitable benefit from the exhibition of some form of suprarenal substance, though in any given case it remains up to now impossible to determine its probable response to the treatment.

(2.) In any given case of the disease, selected haphazard, the probability obtains that disappointment will follow on the institution of organotherapy; but that probability is very distinctly less than that attaching to any alternative method of treatment at present known.

(3.) The last word upon the preparation to be used and its method of administration remains yet to be said. The problem seems to be to get a sufficient and continuous dose of the pure and active principle unchanged into the blood-stream. Intravenous injection is impracticable.

Postscript.—Since the above was in type, Prof. Charles G. Stockton of Buffalo has kindly sent me an interesting note of the subsequent progress of his patient (Case No. 54). "I regret to say," he writes, "that my case followed the usual course. After a period of improvement lasting more than a year, she became suddenly and severely worse, and died within a few days. . . . The autopsy was made by a country practitioner, who informed me that he was unable to find any evidence of either suprarenal body." Dr. Stockton cannot, of course, vouch for the accuracy of the autopsy, but he was under the impression that the glands were in a state of atrophy. The patient suffered also from marked nephroptosis. (Case resembles No. 55.)

Dr. G. Lovell Gulland has also been good enough to inform me that he has used suprarenal preparations in at least half a dozen cases of Addison's disease without being rewarded by a good result.

Recently Hirtz (*Bullet. Gén. de Thérap.* 30 Juillet, 1902) has reported two cases treated by subcutaneous injections of suprarenal extract with very encouraging results. They might be included in *Class III.* In one patient there was progressive amelioration, gain in weight, improvement in the accompanying pulmonary tuberculosis, and a return of strength, so that he was able to assist in ward-work. (March 11—June 17, 1902.) The other patient progressively improved during the six months he was under Hirtz's care. Profound apathy and asthenia were replaced by energy and good spirits. Gain in weight from 52 kg. 700 to 61 kg. The pigmentation cleared up to so remarkable a degree that the patient would scarcely have been taken for an Addisonian. There appears to have been no specific influence on the pulse. The total number of cases referred to in this paper is thus brought up to 105: *Class I.:* 7. *Class II.:* 49. *Class III.:* 33. *Class IV.:* 16.

REFERENCES.

References not here given will be found either in the text or in the list of cases.

[1] *American Journ. Med. Sci.*, July, 1897.
[2] *Lancet*, January 18, 1896.
[3] *Thèse de Paris*, 1896.
[4] Addison: *On Disease of the Suprarenal Capsules*, p. 26.
[5] *Lancet*, June 18, 1898.
[6] *Ibid.*, October 3, 1896.
[7] See Heelas: *Brit. Med. Journ.*, June 8, 1901; Habgood: *Ibid.*, May 25, 1901.
[8] *Dublin Journ. Med. Sci.*, 1882, p. 293, *et seq.*
[9] *On Addison's Disease*, 1875, p. 16, &c.
[10] Abel and Crawford: *Bull. Johns Hop. Hosp.*, July, 1897, and Abel's papers; *ibid*, September October, 1898; November, 1901; February and March, 1902.
[11] See *Therap. Gaz.*, April 15, 1901.

HEADACHE.

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(*Continued.*)

PATHOLOGY.

SINCE headache is no more than a symptom of the many maladies on which it depends, it can possess no special pathology of its own. In certain of its varieties gross lesions are present in the brain, but they and not the headache constitute the disease, and therefore their respective pathological tissue-changes must be sought for in current descriptions applicable to the type of lesion with which, in each case, we are faced. In others, the pathological explanation is to be found in morbid structural alterations in the nose, ear, stomach, uterus, or other organ, the headache being merely a result of sympathetic or reflex irritation. Changes in the vascular system are to be looked for in explanation of some headaches, and morbid conditions affecting individual nerves as responsible for others. Cephalalgia dependent upon ocular causes is without demonstrable change when due to muscular fatigue or refractive error, but it may also be the result of recognisable lesions either in the globe or orbit. Neuropathic headaches, though clinically distinctive, afford no post-mortem testimony to the molecular or other minute departures from normal which must certainly be present, if one could but discover them; and the multitude of toxæmic conditions upon which the large bulk of headaches depend are, in most cases, incapable of demonstration and can only be inferred from reasoning based on analogy, strengthened by experience, and confirmed by the results of treatment, which by counteracting the toxic cause, removes the headache as well as the other symptoms which presumably exist in consequence of its noxious effect upon the organism.

PROGNOSIS.

The prognosis of headache is wholly involved in that of the cause producing it. When this can be ascertained, the prospects of relief or cure will be in direct proportion to the possibility of its removal.

Organic headaches which do not yield to large doses of iodide of potassium, or which are beyond the reach of the surgeon's skill, are prognostically the most grave of all. Migraine may be very intractable, but is generally susceptible of amelioration under the influence of skilful therapeutic management and suitable prophylactic measures. It may, moreover, be foretold of it that, however rebellious it may prove to treatment in early and adult life, it will wear itself out and be recovered from, when the later decades are reached and the fires of vitality begin to burn less brightly. Neuralgic cephalalgia is fortunately amenable to the influence of many analgesic drugs. It usually disappears when the offending tooth or stump is removed, the tympanic disturbance allayed, or the local irritation otherwise, whatever it be, is overcome. The circumstances under which its prognosis is grave are those in which it sets in during later life, without ascertainable cause, in a patient with an inherited tendency to mental disease. In the toxæmic variety the prognosis is widely variable, but must clearly be gauged by the nature of the poison causing the headache. In the acute fevers headache is so subservient to the other manifestations of the disease that its influence as a prognostic item may be disregarded, though it is to be noted that its continuance beyond the usual limits of its endurance should always be considered suggestive of grave and exceptional complications. The headache of rheumatism yields to suitable treatment; so also does that of hepatic disorder, except in those rare conditions where gout is retrocedent and capable of giving rise to meningeal or cerebral complications. The headache of renal disease has always a serious prognostic importance: it may be taken as evidence of failure in the eliminating power of the kidneys, and, in proportion as it is severe and persistent, is ominous of approaching uræmia. The outlook in ocular headaches is, for the most part, favourable. Refractive errors and asthenopic troubles are capable of complete relief by suitable glasses and rest; and most of the organic disorders of the eyeball or orbit upon which they are dependent are within the compass of surgical methods. Neurasthenic headaches are of varying persistence, but the ultimate prognosis is good, if the patient's circumstances are such as to admit of suitable and prolonged treatment. In cases where the headache depends upon hæmic conditions, relief may be promised if the cause lies in excessive dietetic habits which are abstained from, or in anæmic conditions which yield to treatment by iron and arsenic. When it is caused by heart-disease or arterial degeneration, the chances of recovery are more problematical and, for obvious reasons, less to be relied upon.

DIAGNOSIS.

Headache is a very real and prominent fact, and one which obtrudes itself constantly and unpleasantly on the notice of its possessor. It is entirely a subjective phenomenon which is beyond the reach of physical diagnosis, but it is the feature of his illness for which the patient specially seeks relief and, to his mind, it constitutes the central pivot round which the other phenomena of his malady cluster, but to which they are all subordinate. In the large majority of cases the existence of headache must be believed from the patient's testimony alone, and—due latitude being given to the possibility of exaggeration in the description afforded by one who at the moment is suffering more or less acutely—an effort must be made to arrive at an approximately accurate idea of its degree and type by a due consideration of the circumstances under which it has arisen, of the probable cause upon which it depends, and of the lines of evolution which it has followed both in regard to the progress of its own development and its relationship to the other symptoms by which it is attended. The difficulties of differential diagnosis are often considerable and sometimes insuperable, and these are in nowise diminished by the fact that several varieties may be coëxistent or may supervene the one on the top of the other. Although the general fact of headache must be accepted on the patient's statement, its type can only be recognised by an acquaintance with some such description as the preceding, of which the following recapitulation will be found useful.

1. *Organic Headache.*—The pain is usually frontal except in cerebellar lesions, when it is occipital. It is mostly continuous, but varies in intensity, and is as a rule more severe

during the night than during the day. This last characteristic is specially prominent in cases which are of syphilitic origin. There may be localised tenderness of the scalp over the seat of lesion, when this is cortically situated. The pain is apt to be succeeded by convulsions or coma. When it is of inflammatory origin, there is accompanying pyrexia; when it is due to venous thrombosis, there will probably be localised external œdema; and when it is the result of hæmorrhage, it is often limited to one or other parietal region and is rapidly followed by paralysis of a hemiplegic distribution. If caused by a tumour, double optic neuritis and vomiting of cerebral type afford confirmatory evidence, whilst localised palsies are valuable landmarks in the direction of localisation.

2. *Nervous Headache.*—The pain is always at first unilateral, and is most frequently referred to the left temple, whence it may spread over the forehead. It is often accompanied by increase of arterial tension. It occurs in paroxysmal attacks, is preceded by premonitory eye-phenomena, and at its height is sometimes relieved by vomiting. It terminates in troubled sleep after having lasted over a varying number of hours. It is as common in childhood as in adult life, but is rare in old age. A strictly localised type of headache, which occurs preëminently in the chlorotic, is known as "clavus" and belongs to this variety. The painful area can be covered by the tips of two fingers, and is mostly situated in the supra-orbital or temporal region.

3. *Neuralgic Headache.*—The pain is experienced either in one of the three divisions of the trigeminal nerve, when it is referred to the various areas of this nerve's distribution to the face; or in the occipital nerve, when its situation is on the posterior aspect of the cranium. It is of a deep-seated boring character, is always unilateral, and is subject to short acute paroxysmal exacerbations. Tender spots exist at the foramina of the bones whence the branches of nerve emerge. The paroxysmal attacks are often accompanied by spasmodic contractions of the facial muscles. In some cases there may be vertigo, mental confusion, or even delirium.

4. *Toxœmic Headache.*—This is the most common of all varieties of headache and is met with in every form of blood-contamination. Its nature is suggested by the existence of

one or other of the many conditions which are known to be associated with metabolic disturbance, by a history of dietetic over-indulgence, or by the use of certain drugs. The pain occupies the forehead and vertex, and is dull and lasting. When due to alcoholic or dietetic excess, it is usually worst in the morning, and is speedily relieved by vomiting. A sudden attack of frontal headache, accompanied by vertigo, slight delirium, increased arterial tension, and changes in the urinary secretion, suggests the retrocedent form of gout, when occurring in those who possess the gouty diathesis. Acute superficial pain over the forehead and occiput, with accompanying tenderness of scalp, is characteristic of rheumatism, especially if it sets in after exposure to cold or damp in a person who has previously suffered from other forms of rheumatic affection. When the headache results from hepatic disorder, the liver is full and tender; there are marked mental depression, a muddy or icteric condition of skin, and the usual changes in urine and bowel-evacuations which result from a deficiency of bile.

When it is the consequence of renal disease, the pain is continuous and is accompanied by a sense of fulness in the head, ringing in the ears, dimness of sight, increased arterial tension, breathlessness on exertion, a tendency to delirious excitement and subsequent drowsiness, vomiting or diarrhœa, and often diminution in the amount of the urinary secretion. Retinal changes are sometimes present, and analysis of the urine reveals the presence of albumen and other abnormalities.

5. *Ocular Headache.*—The pain is often complained of in the occipital region, but may be entirely referred to the eyeballs and orbits. It comes on after prolonged use of the eyes, and is associated with blurring of vision, hyperæmia of the conjunctivæ, and congestion of the lids. It is relieved by rest, and in refractive cases is ended by the use of suitable spectacles. When due to glaucoma or intra-ocular tumour, there is increase of ocular tension and the ophthalmoscope reveals the reason.

6. *Neurasthenic Headache.*—The pain may be superficial or deep, is mostly occipital, but sometimes vertical in situation, is accompanied by a sensation of constriction as though the head were bound round with an iron band, and is frequently complicated by intercurrent attacks of true migraine. It is markedly increased by light or noise, and produces general

hyperæsthesia of the scalp. Emotional instability and vasomotor phenomena are constant concomitants. It constitutes part of the general picture of neurasthenia, and disappears under the influence of rest and relief from work and worry.

7. *Hœmic Headache.*—There are two types of this headache. The plethoric occurs in full-blooded persons, and causes a dull pain, extending from the forehead to the occiput, which is increased by effort, mental excitement, or stooping, and relieved by complete rest in a semi-upright position. The anæmic occurs in pallid, bloodless patients, and causes pain mostly referred to the forehead or vertex, accompanied by palpitation, throbbing in the head, dizziness and a tendency to syncope. It is relieved by pleasurable excitement or a stimulating meal, but is intensified by undue exertion or the upright position.

TREATMENT.

In a subject embracing such wide and diverse considerations as headache, the question of treatment can only be partially dealt with. An endeavour will here be made to sketch a plan of management suitable to each of the varieties of the disorder which have been considered, but no more can be claimed for it than expediency. No one plan will be invariably successful, and the following recommendations must be accepted with the reservation that they are no more than the outcome of personal, and therefore limited, experience, and that they claim only to be suggestive and not by any means inclusive. The reader must fill in the gaps from his own knowledge of therapeutics and by reference to special treatises.

The first essential in successful treatment is to diagnose the case aright and, if possible, to remove the cause upon which the headache depends. It is on account of this primary need that some scheme of classification is necessary, because, however partial and incomprehensive such a scheme may be, it at least serves to marshal the facts which we possess in some measure of methodical arrangement, and enables us to guide whatever treatment we adopt on lines of actual knowledge or reasonable hypothesis. This plan is assuredly more likely to be attended by satisfactory results than any mere haphazard attempt to deal with headache as something *per se*, or as a condition to be handled irrespective of its ætiological relationships or collateral manifestations. In all cases of headache, causes of peripheral irritation should be first sought for by a careful investigation of the scalp, the eyes, the nasal cavities, the auditory apparatus, the mouth, and the throat. The state of the digestive organs ought to be next enquired into, and thereafter the condition of the blood and of the vascular and nervous system must be ascertained. The other organs of the body demand no less careful attention in our search after an adequate cause, and particularly the liver, kidneys, and brain must be looked to. The possibility of a gouty, rheumatic, or syphilitic diathesis being responsible for the headache should not be forgotten, and, finally, the habits of the patient, together with the state of his environment from the hygienic point of view, must be brought within the scope of our investigation. Of mental causes upon which headache may depend, the most prolific is worry, which wears out the nervous system by keeping it in a perpetual condition of strain. The temperament that is irritable and prone constantly to worry, often over trifles, ought to be schooled into a more philosophical view of life and its vicissitudes, if the risk of ultimate annoyance from headaches is to be avoided, or the cure of them, when once they are established, to be accomplished. Intellectual work, properly adjusted, is conducive to robustness of health, both mental and bodily, but if it is pushed beyond the limits of toleration, especially in a growing boy or girl, it is liable to impair the constitutional vigour and to be provocative of headache as part of the penalty.

1. *Organic Headache.*—When the pain is the result of meningitis, sinus-thrombosis, or hæmorrhage, each of these conditions must be treated according to the directions given for its management in the text-books of medicine and therapeutics; but when there is reason to fear, from the nature of the cephalalgia and the accompanying symptoms, that an intracranial growth is the root of the evil, a clear course of procedure is indicated. In all such cases iodide of potassium must be relied upon as the drug likely to afford relief if any drug will. If the tumour depends on syphilis, the iodide may prove entirely curative; but it also often relieves pain and local congestion induced by non-specific swellings. The important point to be noted in regard to its administration in such cases is

that to be efficient the doses must be large, from 30 to 40 or even 60 grains. In the presence of constitutional syphilis, iodism is seldom produced by these large quantities, and the risk may be, to some extent, provided against by combination with ammonia or small doses of arsenic. The following formula is convenient :—Iodide of potassium, 40 grains ; aromatic spirit of ammonia, ½ a drachm ; Fowler's solution, 2 minims ; decoction of bark, ½ an ounce : to be given at first every four hours, lessening to every six, and then to every eight hours as relief is obtained. At first it may be necessary to secure sleep by full doses of chloral hydrate, or of antipyrine combined with bromide of potassium ; but if these fail, as they often do, hypodermic injections of ¼ or ⅓ of a grain of morphine must be resorted to at bedtime until the iodide begins to take effect. Local measures, such as blisters to the temples or counter-irritation to the nape of the neck, are of little value. If medicinal measures fail and the tumour is within reach, the question of surgical interference must always be considered.

The patient should be kept entirely in the recumbent position in a quiet and darkened room ; his food should consist of fluid or semi-fluid nourishment administered in small and frequently-repeated quantities, and the bowels must be freely evacuated daily. An ice-cap to the head often affords some relief, and if there be much sickness, ice may be given to suck and a mustard-plaster occasionally applied to the epigastrium.

2. *Nervous Headache.*—The remedies which have been advocated for the relief of this variety of headache are without number. There seems to be no absolute rule to explain why in one case a certain drug affords speedy relief, whilst in another of apparently similar type it is entirely useless. The most that can be expected from any analgesic remedy is that it may lessen the severity and duration of the pain. The wave-storm once started must blow itself out, and can very seldom be immediately and completely arrested after it has fairly begun to rage. The important point in the treatment of these cases is so to fortify the patient's general health by correcting unhygienic conditions and pernicious habits that he acquires a greater resisting-power from improvement in the stability of his nervous centres and greater physiological activity in the secretory and excretory functions of his various organs.

When a headache is threatening the patient should at once give in to it, and devote himself to complete rest and seclusion. If the onset of pain can be referred to immediately-preceding fatigue, a glass of champagne or a small tumblerful of brandy and soda-water, together with ten grains of antipyrine, will often succeed in relieving it. This should be followed by an active aperient given at night, and none will be found better than a pill consisting of 1½ grains of calomel and 3½ grains of compound colocynth mass; followed in the morning, if need be, by a full dose of Carlsbad salts, Rubinat water, or other simple saline. But in cases that are at all severe, such simple measures are unavailing; the stimulant may indeed even aggravate the discomfort. It is then necessary to put the patient at once to bed in a darkened room. Cold applied to the head by means of an ice-bag, or cloths wrung out of iced water, or Leiter's tubes, is often grateful, and the severity of the pain is always lessened by the application of a mustard-plaster down the spine. The plaster should be long enough to extend from the roots of the hair to the sacrum, and wide enough to cover the seat of exit of the spinal nerves; it ought to be kept on while it can be comfortably borne, and this must be at least long enough thoroughly to redden the skin below it. During the continuance of the pain, no aliment of any kind need be given. When there is reason to believe that the stomach contains a quantity of undigested and fermenting food, this should be removed either by means of the syphon-tube, or through the agency of an emetic dose of sulphate of zinc or ipecacuanha. The variety of pain-killing drugs with which the market is flooded makes it difficult to decide which is most to be relied on, but probably the three which are of greatest value are antifebrine, antipyrine, and exalgine. Combined as follows, one or other of those will often prove effective :—

(1) Antifebrine, 5 grains; caffeine, 5 grains: to be put up in a cachet and taken every two hours for three doses or until the pain subsides.

(2) Antipyrine, 15 grains; salicylate of soda, 15 grains; spirits of chloroform, ½ a drachm; water to ½ an ounce: to be taken every two hours for three doses, thereafter every four hours.

(3) Exalgine, 2 grains; bromide of sodium, 20 grains; tincture of orange, 20 minims; water to ½ an ounce: to be taken every two hours for three doses, thereafter every four hours.

In cases where all the above formulæ fail to give relief, such other remedies as phenacetin, croton-chloralhydrate, guarana, antitoxine, &c. may be tried. It may even be necessary, in extreme cases, to resort to morphine hypodermically, but this should only be done after every other means of relief is exhausted on account of the danger of begetting a morphia-habit. This danger is probably exaggerated by most writers, and the relief afforded is so instantaneous that it may be disregarded in cases where other measures have proved unavailing. It is most efficacious when given in combination with atropia; ¼ of a grain of morphine with $\frac{1}{100}$ of a grain of atropine being usually sufficient to allay the pain.

When the migrainous attack is accompanied by a pulse of high tension, each dose of whatever remedy is chosen should be accompanied by a tablet containing one minim of a one-per-cent. solution of nitro-glycerine. Between the attacks of pain —in addition to the proper regulation of the daily life as regards diet, exercise, clothing, work, and rest—brilliant results in the way of prevention are sometimes attained by the recognition and correction of the cause on which they depend.

Of the various specific drugs that have been found beneficial, apart from those which may be required for the correction of this, that, or the other dyscrasia, none seem to be attended by better results than arsenic and cannabis indica. To these iron may be added with advantage in many cases, as in the following combination:—Reduced iron, 3 grains; arseniate of soda, $\frac{1}{80}$ of a grain; extract of cannabis indica, ⅓ of a grain; extract of rhubarb, ¾ of a grain: to be made into a pill; one to be taken after meals three times a day for three weeks out of four during from four to six months. After a time, when toleration is established, the dose of both arsenic and Indian hemp ought to be increased.

In several recent cases the substitution of ¼ of a grain of cacodylate of soda for the arseniate of soda, seems to the writer to have produced a more decided effect, presumably because of the larger quantity of arsenic which it is possible to administer in this way. Its objection is the powerful garlic-like odour which, after a few days, the cacodylate communicates to the breath.

The bromides possess a special efficacy in diminishing the excitability of cortical cells and regulating the cerebral circulation. A single 30-grain dose of one or other of the salts, or a combination of all three of them in equal proportions, taken at bedtime, may be ordered with such a tonic as that suggested. Bromide is specially indicated when the rest at night is broken or disturbed by frightsome dreams.

Setons inserted into the nape of the neck and worn for three or four months are said to be prophylactically valuable, but their discomfort is a strong argument against their adoption.

3. *Neuralgic Headache.*—In this, as in all other forms of headache, the first indication is to remove the cause, when it can be discovered and is capable of removal. Such conspicuously rational treatment in no way interferes with the various methods which may be adopted for the relief of existing pain, particularly since the implicated nerve may have been so influenced as to induce a continuance of the neuralgic pain for some time after the exciting cause is removed. Anæmic, gouty, rheumatic, malarial, or syphilitic antecedents must be counteracted by iron, colchicum, salicylate of soda, quinine, iodide of potassium, or mercury, according to the indications afforded. Errors in nutrition should be corrected by suitable dieting, regulated exercise, massage, &c. The bowels must be regulated by mild aperients, to which $\frac{1}{8}$ of a grain of podophyllin, or 2 grains of grey powder, may be added when there is evidence of hepatic sluggishness. In women the menstrual function, if irregular, demands attention. Local sources of irritation in the ear, mouth, or throat should be sought for, and particularly the teeth must be carefully examined in order to be sure that a buried stump is not primarily responsible for the pain. The various antra of the facial bones also require investigation. Locally, the application of sedatives often succeeds in relieving the intensity of the suffering, particularly at the points of nerve-exit from the skull where it is usually most severe. Menthol is specially useful for this purpose and may be combined with chloroform as follows :—menthol, 3 drachms ; pure chloroform, 2 drachms ; linimentum saponis,

11 drachms; to be gently rubbed over the painful area, and the part to be afterwards covered with warm cotton-wool. Equal parts of extract of belladonna and treacle spread on lint and applied over the tender nerve under a hot linseed-poultice, or spongiopiline wrung out of boiling water; or a strong solution of atropine, 5 grains to the ounce, rubbed with the finger into the painful part, are also efficacious local remedies. The use of the continuous current is sometimes attended by gratifying results.

During the paroxysms, the treatment of the pain demands prompt and active measures. In all cases where it is very acute, initial relief should be obtained by one or more subcutaneous injections of either morphine or cocaine, in doses of ¼ of a grain. Either of these drugs may, after one or two administrations, prove curative, but when this is not the case, its influence must be helped by the exhibition of other remedies. Many of the analgesic drugs which have been referred to as useful in nervous headache, are equally valuable in this variety of the disorder, but there is one drug which more than any other seems to exercise a specific effect upon peripheral nerves, more especially the trigeminal branches, and that is Gelseminum Sempervirens. It may be given in either liquid or solid form, and ought to enter into the composition of any mixture designed for the relief of headache of the neuralgic type. From the following formulæ, that one should be chosen which seems best to meet the indications of the case on hand:—

(1) Ammoniated citrate of iron, 5 grains; tincture of gelseminum, 15 minims; Fowler's solution, 5 minims; liquor ammoniæ acetatis, 1 drachm; peppermint-water to 1 ounce: to be taken three times a day, after meals.

(2) Citrate of potash, 15 grains; piperazine, 5 grains; compound tincture of colchicum, 15 minims; tincture of gelseminum, 15 minims; chloroform-water to 1 ounce: to be taken every six hours.

(3) Salicylate of soda, 10 grains; tincture of actæa racemosa, ½ drachm; tincture of gelseminum, 15 minims; camphor-water to 1 ounce: to be taken every six hours.

(4) Salicylate of quinine, 2 grains; extract of gelseminum, ¼ of a grain; extract of cannabis indica, ¼ of a grain; extract

of henbane, 1 grain: to be made into a pill; one to be taken every six hours.

(5) Iodide of potassium, 10 grains; tincture of gelseminum, 15 minims; carbonate of ammonia, 3 grains; compound decoction of sarsaparilla to ½ an ounce. To be taken after food, three times a day.

When there is no dyscrasial indication, the following pill will often be found efficacious:—Croton-chloral hydrate, 5 grains; extract of gelseminum, ¼ of a grain; extract of cannabis indica, ⅓rd of a grain; extract of gentian, q.s. To be made into a pill and taken every three hours until relief is obtained.

In cases of unusual severity, especially such as occur in a patient past middle life with an inherited tendency to mental disease, none of the above drugs may be successful. Relief may then sometimes be obtained from hyoscine, which should be given in pill-form, in doses of $\frac{1}{100}$ of a grain, twice or three times a day.

When all drugs fail, the surgeon's services may be requisitioned with a view to stretching the nerve, or to the removal of the Gasserian ganglion.

After the pain is relieved, some attempt should be made to improve the general tone of the system by tonics, codliver-oil, abundant food, and change of air.

4. *Toxæmic Headache.*—The form of toxæmic headache which is induced by drugs, alcohol, or tobacco can only be cured by the withdrawal of the poisonous substance which is responsible for its production. When the headache arises from pyrexia consequent upon specific fevers or inflammatory lesions, it is generally confined to the earlier stages of the illness and will yield in time to the effect of the remedies administered for the relief of the disease on which it depends. It is, in most cases, allayed by cold affusion to the scalp, but otherwise its treatment becomes merged in that of the general disorder. When it results from the breathing of impure air, rest in the recumbent position and ten grains of phenacetin or antipyrine in half an ounce of brandy will speedily set it right. This ought to be followed by a smart purgative dose taken at bedtime.

The headache which occurs with influenza is relieved by the following mixture: — Salicylate of soda, 15 grains; antipyrine, 10 grains; liquor ammoniæ acetatis, 1 drachm; camphor-water to an ounce: to be taken every two hours for three or four doses, thereafter every four hours. In enteric fever, headache is often so severe as to demand special treatment, but it does not yield to analgesic drugs in the same satisfactory way as the headache of influenza. Probably the most reliable remedies are chloral hydrate and bromide of potassium, which may be given thus:—Chloral hydrate, 10 grains; bromide of potassium, 20 grains; compound tincture of lavender, 20 minims; glycerine, ½ a drachm; water to ½ an ounce: to be taken every three or four hours.

The headache which is associated with imperfect digestion is due primarily to two important conditions: 1st, to arrest or delay of the hepatic function; and 2nd, to fermentative or putrefactive processes in the gastro-intestinal tract. For the relief of the former condition, one or other of the following pills should be given every night, or every alternate night according to the result, until six are taken.

(1.) Podophyllin, ¼ of a grain; extract of rhubarb, 4 grains; extract of belladonna, ⅙th of a grain; confection of roses, q.s.

(2.) Pil: hydrargyri, 1½ grains; extract of colocynth, 2½ grains; gingerin, ¼ of a grain; extract of henbane, ¾ of a grain.

(3.) Iridin, 2 grains; jalapin, 1 grain; capsicin, ⅙ of a grain; extract of rhubarb, 1½ grains.

If necessary, the action of the pill may be assisted by a morning dose of some simple saline.

For the prevention of intestinal fermentation, antiseptics will be found useful, and may conveniently be given either in acid or alkaline media according to the indications of the case.

(1.) Dilute hydrochloric acid, 15 minims; pure carbolic acid, 2 grains; strychnine-solution, 5 minims; tincture of ginger, 20 minims; decoction of bark to 1 ounce: to be taken three times a day one hour after each meal.

(2.) Sulphocarbolate of soda, 15 grains; bicarbonate of soda, 15 grains; tincture of nux vomica, 10 minims; spirit of chloroform, ½ a drachm; compound infusion of gentian to 1 ounce: to be taken three times a day between meals.

The food ought to be of the simplest description and carefully adapted to the patient's digestive capacity. When the stomach is flabby or dilated, it should be washed out through a syphon-tube every morning. In cases where the headache is the result of a large and unsuitable meal or of an overnight debauch, an emetic is the speediest means of relief. The headache which is associated with gout may be expected to yield along with the other symptoms when the excess of uric acid in the blood is counteracted by the use of suitable remedies, but in that form of sudden and acute pain which either interrupts the normal progress of a gouty inflammation of the great toe, or seems to come in place of it, measures more actively directed to the head-condition are demanded. In addition to the usual anti-gouty remedies, an attempt should be made by means of counter-irritation to the great toe to induce a return to it of the inflammatory process. The head should be kept cool, and if the pulse-tension be raised, four or six leeches may, without hesitation, be applied to the temples. The bowels ought also to be copiously evacuated by a 5-grain dose of calomel given at night, and followed next morning by ½ an ounce of Epsom salts in a claret-glassful of water, repeated every hour until a result is obtained.

The headache of rheumatism is best relieved by the local application of warmth, and often disappears speedily under the influence of this powder:—Chloride of ammonium, 20 grains; salicin, 10 grains; phenacetin, 10 grains: to be shaken up with water and taken every six hours. In the headache due to renal change, the patient should be put on a strict milk-diet, and the action of the skin, kidneys and bowels stimulated by diaphoretic, diuretic and purgative drugs. The best of all diuretics is pilocarpine, which may be given hypodermically in a daily dose of ¼ of a grain, the patient being at the same time placed in a hot pack, where he should remain for an hour. The ordinary diuretics, such as citrate or acetate of potash, squills, digitalis, spirit of juniper, or cream of tartar, often best serve the purpose of promoting a free action of the kidneys; but when they fail, success may follow the administration of diuretin in 10-grain doses every four hours. Hot fomentations or poultices over the loins promote the action of whatever diuretic drugs are given. The purgative chosen should be an active one, such as compound jalap powder in doses of 40 to 60 grains, or croton-oil in the form of such a pill as the following:—croton-oil, ½ a minim; caraway oil, 1 minim; extract of colocynth, 4 grains. When the headache is accompanied by high arterial tension, oppression in the chest, and difficulty of breathing, it will often be relieved by this mixture:—iodide of potassium, 10 grains; solution of nitroglycerine, 1 minim; liquor ammoniæ acetatis, 1 drachm; aromatic spirit of ammonia, ½ a drachm; infusion of calumba to 1 ounce: to be taken every four hours.

5. *Ocular Headache.*—The common form of this variety of headache disappears under the influence of complete rest of the eyes, and is cured by the use of glasses properly adapted to correct existing errors of refraction. When it results from organic changes in the eyeball or orbit the services of the ophthalmic surgeon must be sought for its relief. Simple asthenopia demands, in addition to rest of eyes, tonics suited to the circumstances of the case.

6. *Neurasthenic Headache.*—The treatment of neurasthenic headache is practically comprised in that of the toxæmic variety. Neurasthenic manisfestations are probably due in the first place to a faulty metabolism, whereby fermentative processes are encouraged and toxines elaborated which, because of the unstable condition of the nervous system, act, even in small amount, with undue virulence. The general line of treatment which has been suggested as useful in toxæmic conditions will, therefore, also apply here, but there is one formula specially suitable to neurasthenic cases which may be added as an alternative to those already mentioned, or rather as an addition by means of which the improvement produced by them may be firmly established:—Valerianate of zinc, 3 grains; strychnine, $\frac{1}{80}$ of a grain; carbolic acid, 2 grains; extract of cannabis indica, ⅓ of a grain; extract of rhubarb, ½ a grain: to be put into a capsule and taken after meals three times a day.

In neurasthenic cases which are accompanied by much disturbance of the vasomotor system, such as is so often met with at the climacteric in women, the value of ichthyol should not be lost sight of. Under such circumstances it should be substituted for the carbolic acid in the foregoing formula; 5 grains being, in most cases, a sufficient dose. There is probably no drug in present use which may be more depended upon to steady the circulation and restore its stability. Whether this is accomplished by a direct effect upon the nervous system, or by a secondary antiseptic influence on the gastro-intestinal canal, there is no present evidence to prove. It can, however, hardly be altogether the latter, because it frequently succeeds when carbolic acid has entirely failed to effect any improvement.

7. *Hæmic Headache.*—In this variety of headache the treatment will vary according as the pain is the result of a plethoric or of an anæmic state of the brain. In plethoric conditions the crying need is to reduce the cerebral fulness. This is to be effected by abstinence from alcohol, and by a dietary wholly confined to milk and fish. The bowels must be freely acted upon by a saline purgative in the early morning, and by an occasional pill, such as already quoted, containing mercury or podophyllin, given at bedtime. If the urine is scanty and loaded with urates, diluents should be taken abundantly, and, in addition, a simple alkaline mixture of this type :—Bicarbonate of potash, 20 grains ; nitrate of potash, 10 grains ; antimonial wine, 10 minims ; juice of taraxacum, ½ drachm ; carraway water to 1 ounce : to be taken every four hours. When there is a gouty element in the case, the wine or tincture of colchicum in 15-minim doses should be added to this mixture ; and if there is undue cerebral excitement, the bicarbonate should be replaced by the bromide of potash in a similar dose. Cold should be applied to the scalp, and, if the patient is full-blooded and vigorous, it may be desirable to reduce the volume of the blood either by venesection or leeching. Temporary relief is always obtained by a hot mustard footbath, or by a large linseed-and-mustard poultice applied over the abdominal wall. Sleep is often seriously interfered with, but is generally restored by 15 grains of trional or sulphonal given at bedtime. When the acute phases of this congestive headache are overcome, the vessels of the brain may often be kept contracted under the influence of ergot and hamamelis :—Ergotin, 1 grain ; hamamelin, ½ grain ; extract of colchicum, ½ grain ; euonymin, 1 grain : to be made into a pill and taken three times a day. If this form of headache is the result of overwork, the mind must have entire relaxation, and a period of complete rest either at the seaside or at a suitable foreign watering-place is much to be desired.

As regards the treatment of the anæmic form of headache, much will depend upon the special symptoms of each individual case. The cause of the anæmia must be ascertained, and removed if possible. Discharges of all kinds demand prompt attention, and derangement in the assimilative functions should be corrected. All remedies which increase vascular tension and accelerate the flow of blood through the brain are serviceable. Of these, the most important are arsenic and iron. These are capable of combination in almost endless variety, but the two following formulæ will be found to meet the requirements of a large majority of cases, the first to be given at the commencement of treatment and followed, after a time, by the second :—

(1) Ammoniated citrate of iron, 7 grains ; Fowler's solution, 5 minims ; liquor ammoniæ acetatis, 1 drachm ; bromide of ammonium, 10 grains ; chloroform-water to an ounce : to be taken three times a day, after meals.

(2) Sulphate of iron, 3 grains ; arsenious acid, $\frac{1}{40}$ of a grain ; sulphate of quinine, $\frac{3}{4}$ of a grain ; powdered digitalis, $\frac{1}{2}$ grain ; extract of camomile, q.s. : to be made into a pill and taken three times a day, after meals.

When there is sleeplessness at night, opium ought to be given in small doses. In the form of Dover's powder and combined with rhubarb, 5 grains of each, it meets the requirements of many cases. It is best given in a cachet at bedtime. Anæmic headaches are relieved by the moderate use of stimulants, and in the majority of cases a light claret or Burgundy with luncheon and dinner suits well. The relief furnished by alcohol to the misery of cerebral anæmia makes it one of the most fruitful sources of drinking-habits, especially among women ; it must, therefore, always be ordered with a strict regard to moderation, and ought only to be allowed with meals. Every case of anæmic headache is benefited by change of scene and pleasant society, both of which tend to dissipate the mental depression which is such an unfailing accompaniment of an impoverished condition of the blood.

This attempt to focus the far-reaching subject of headache within the limits of a description that will embrace its main features and also indicate a line of treatment from which good results may be anticipated, is painfully imperfect. It seems but to emphasise how desultory is our knowledge of a disorder which, though only a symptom, is yet so universal and embraces such an enormous field of human misery that the doctor is daily called upon to puzzle himself over its nature and cause, whilst the patient regards it as so much the crowning sorrow of his condition that he too often blames unfairly the skill of his physician and the resources of medicine, because both alike are sometimes unable to afford him the relief he so urgently requires. In these days of hurry and rush, the causes which may produce headache are becoming rapidly multiplied, and since the foundations of future cephalalgic troubles are frequently laid in the nursery and in the schoolroom, it behoves everyone who is concerned with the management of the young so to regulate their habits of work and recreation, of rest and exercise, as to ensure, as far as that is possible, a jealous regard for the integrity of those delicate nervous structures which are the seat of life and energy, and on whose stability future health and happiness so entirely depend. "In bringing up a child, think of its old age" is a council of perfection which, from every point of view, but in the present relationship particularly from the headache point of view, is worthy of the fullest acceptation.

ON THE FREQUENCY OF ALIMENTARY GLYCOSURIA IN THE CHILDREN OF DIABETIC PERSONS.

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DIABETES is, according to the notion of the day, a disease of which the principal symptom consists in a diminished or absolutely abolished ability to assimilate carbohydrate food.

Any normal individual can ingest an almost unlimited quantity of starchy food without passing a trace of sugar in his urine that could be found by the ordinary tests of Trommer, Nylander, &c. It is otherwise with the diabetic. He will eliminate a smaller or larger quantity of sugar according to the quantity of amylaceous food which he has introduced into his system.

We must not think, and it would be also contrary to the elements of pathology to presume, that such a dystrophy could be the result of a sudden change in the ordinary metabolism of the sugar. It has certainly been prepared insidiously by a long course of events. It has been slumbering in the diabetic when he was still a child; then owing to an irrational nourishment, probably aided by nervous influences, psychical emotions, grief, sorrow, &c., it breaks out sooner or later into open diabetes. Its first manifestation may be the presence of traces of sugar (alimentary glycosuria *e saccharo*) after the ingestion of a quantity of grape-sugar (exceeding 50 grammes); the next degree would be the elimination of traces of sugar after starchy food (alimentary glycosuria *ex amylo*); the higher degree of this pathological state is the "light" diabetes, which, owing to indiscretion in diet, or, what is still worse, to a long-continued irrational ("too severe" *à la Cantani*) dieting, can pass into the "severe" diabetes with its following acid intoxication (Stadelmann) and final coma. This is the general course of the disease, and exceptions, for the better or for the worse, are frequent.

As a result of such considerations, and given the fact that diabetes very often develops itself on a hereditary base, it is very easy to suppose that what is present in the diabetic parent to a larger extent will also show itself to a greater or smaller extent in his child. To ascertain this I have given to several children of diabetic parents amylaceous test-dinners with a view to produce alimentary glycosuria *ex amylo.* I have attributed to this pathological state a greater importance for the purpose of my deductions, as it is in closer relations with the real diabetes, and is not so dependent upon individual influences as the alimentary glycosuria *e saccharo.* According to v. Noorden[1] all persons who eliminate sugar after starchy food must be suspected of real diabetes, and need careful watching. I have given the following test-dinner:—Barley-soup, beef or a thin cutlet, a plate of potatoes and one of rice, a piece of cake, a few figs; half a litre of Bavarian beer, two rolls of white bread, equivalent to a total amount of about 250 to 300 grammes of carbo-hydrates. I have preferred to give such mixed test-meals, as they are more apt to cause glycosuria, as was shown by Kleen[2] in his interesting work on diabetes. The urine that has been eliminated between two and four hours after these test-dinners has been examined by the tests of Trommer and Nylander, and in doubtful cases by the phenylhydrazin test of Fischer with the modification of v. Jaksch, and by the fermentation test. These investigations have been performed on ten persons who were in a healthy condition; two of them, however, presented symptoms of neurasthenia, a state in which alimentary glycosuria is not infrequently found. I may point out, however, that nervous diseases and diabetes often occur in the same family. I have taken pains to secure that no drugs were administered at the same time, such as morphia, salicylate of sodium, &c., which could have accounted for a possible glycosuria. In the following lines I will give a short history of the cases with the result of the examination by test-meals:—

Case 1.—A. N., a boy of 11 years. His mother, 48 years of age, suffering of severe diabetes, mentioned that her mother, too, used to be very thirsty in the latter years of her life (she died at the age 68). The urine after an ordinary meal strongly reduced Trommer's test, and the reducing substance was ascertained by the phenylhydrazin test to be grape-sugar. After the test-dinner there has been found 0·1 per cent. of sugar. The patient suffered from acne of the face and small furuncles in the neck. He used to get up in the night to urinate. He has a sister ot 14 who is getting unduly fat. He appears to be a nervous boy.

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[1] v. Noorden: *Die Zuckerkrankheit*, 3rd edit., Berlin, 1901, p. 15.

[2] Kleen: *Diabetes mellitus and Glycosuria*, 1901, p. 18.

Case 2.—Miss H., 18 years old, was in vigorous health, but she had a tendency to obesity. Her father had light diabetes at 60 years of age. Her urine from the night till morning, examined on two occasions, showed at the first examination small traces of sugar, but none at the second. A test-dinner, twice given, was each time followed by the appearance of distinct traces of sugar in the urine. She used to have frequent outbreaks of acne and sometimes furuncles.

Case 3.—J. H., brother of the last case, is 16 years old. His morning water contained no sugar, but after a test-dinner there were found small traces of sugar.

Case 4.—E. D., a girl of 12 years, was in robust health, but with adenoid vegetations. She exhibited many comedones in face. Her mother died of severe diabetes. Her urine showed no trace of sugar after a test-dinner. My colleague, Dr. Müller, of Carlsbad, having removed the adenoid growths, the urine has again been examined on the day following the operation after administration of a test-dinner, but no sugar was found. A sister of her mother was following the Carlsbad cure for obesity (weight 125 kilos.). At the beginning of her case I have found small traces of sugar in the morning urine, somewhat more after an ordinary meal, and very distinct traces after a test-dinner.

Case 5.—A brother of the last case, 13 years old, used often to have acne of the face and little furuncles on the neck. After a test-dinner distinct traces of sugar were found in the urine at two examinations. He exhibited also a few spots of grey hair.

Case 6.—Mrs. L., 24. Her mother, aged 52 years, is under my treatment for light diabetes. The water examined after a test-dinner showed in the case of Mrs. L. no trace of sugar. She has a tendency to obesity.

Case 7.—Mr. S., brother of the last, aged 22 years. His water after a test-dinner contains distinct traces of sugar.

Case 8.—Dr. H., aged 35 years, suffers from neurasthenia. His mother had light diabetes, suffered for a long time from Pruritus vulvæ, and died in consequence of gangrene. As a medical student he discovered by Trommer's test the presence of some strongly-reducing substance in his urine, which he thought was sugar. Since then he ascertained that each time he has eaten rice or sweet food he eliminates traces of sugar in his water. On taking 100 gr. grape-sugar two hours after a light breakfast he passed 0·1 per cent. of sugar. When a boy his face was covered with acne-spots, and even now a few such lesions often appear on his face and neck. Impotency would show itself temporarily. He exhibits a tendency to fatness, and has many spots of grey hair.

Case 9.—Mrs. T. M. is 40 years old. Her father had diabetes. Her urine contains distinct traces of sugar after a test-dinner. She suffers from neurasthenia. Her hair is almost entirely grey.

Case 10.—Mrs. F., 26 years old, mother of three children, was following the Carlsbad cure three years ago, having passed gall-stones several months before. All that time I found traces of sugar in the afternoon urine, which I attributed to accidental transitory glycosuria. I was, however, more sure of the nature of her glycosuria when her mother (65 years old) came to Carlsbad this summer to follow a "cure" for obesity and light diabetes, which, as seems to be usually the case, was found accidentally last winter by the local practitioner.

As we have seen in the history of these cases, in 8 cases out of 10 alimentary glycosuria *ex amylo* was found. I may mention here that according to the lately-published work of Hirschfeld,[1] normal urine should not contain more than 0·05 per cent. of sugar, which cannot be found by the ordinary tests (Trommer, Nylander).

It seems accordingly that in the majority of the cases the children of diabetic persons have inherited a certain defective power of dealing with sugar in their economy. Apparently in such persons the power to assimilate carbo-hydrates is diminished. This is the principal symptom of an inherited diabetic predisposition. I may mention here shortly that there are other important symptoms too. Thus I have very frequently found in such persons the presence of acne vulgaris not only in the years of puberty, but also in fully grown-up persons. Several of the patients whom I mentioned above had acne. Obesity was also frequent amongst them, and according to v. Noorden[1] this is a symptom of diabetic predisposition. Such persons have lost the ability to burn thoroughly the sugar in their organisms, but they can still deposit it in the form of fat in the tissues. v. Noorden calls it "diabetogenic" fat.

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[1] Hirschfeld: *Die Zuckerkrankheit*, Leipzig, 1902, p. 12.

It is also noteworthy that precocious puberty can often be observed in such children. They very often present a healthy red colour, and are frequently of a bright and active disposition. They not seldom get short-sighted after the years of puberty, and they also often get grey before the usual time. Gingivitis, alveolar pyorrhœa, atrophy of the gum, and retraction of the dental pulp also constitute symptoms of a diabetic predisposition that can frequently be found in descendants of diabetic parents. Senile changes, as atrophy of the gum and retraction of the pulp, can be found in such individuals even when they are at a comparatively young age.

All these persons are candidates for diabetes, but it does not necessarily follow that the disease will develop in each case. It can develop itself in persons who are addicted for a long time to an irrational nourishment (containing abundance of meat and also of sweet and amylaceous food). Its appearance in the majority of cases is about the 35th to 40th years. Owing to great psychical emotions, however (grief, sorrow, and especially psychical shock), it may appear much sooner. Of course, the prognosis will be better the later, and worse the sooner, diabetes actually makes its appearance. And this will show us the line of conduct to adopt. We must do our best to defer its advent to a late period, or, if it is possible, to prevent its onset altogether. For this purpose we should recommend such persons not to take any great quantity of sweet or amylaceous food. We should also advise them to lead rather a frugal than a luxurious life, avoiding rich food, as this would increase the morbid tendency, especially if for a long time much meat is taken simultaneously with large quantities of sweet and amylaceous food. I refer here to the well-known fact that in India diabetes is exceedingly rare among the poor

[1] v. Noorden: *Die Zuckerkrankheit*, 3rd edit., 1902, p. 55.

Hindus, who live only on rice, and most frequent among the rich Hindus, who eat much meat as well as rice. But in Europe also we may observe the fact that diabetes is most frequent amongst classes who eat much meat and amylaceous food simultaneously. Thus it is more frequent amongst the rich than the poor.

It is an interesting fact that the extirpation of the pancreas produces diabetes only in those birds which eat meat (birds of prey), whereas geese, ducks, pigeons, &c., as a rule do not show diabetes after the extirpation of the pancreas. (Minkowski, Weintraud, Kausch.)

As it is known, the products of decomposition of the meat produce a hyperactivity of the thyroid gland, and, as I have established in a communication[1] to the Congress of Madrid, the hyperactivity of the thyroid gland can produce diabetes, especially when there are simultaneously degenerative changes in the pancreas (islands of Langerhans).

We must equally supervise the psychical education of these children. We should recommend them not to embrace callings in which they might be exposed to great intellectual strains or psychical emotions (physicians, lawyers, stockbrokers). On the other hand I consider sport of all kind very valuable for such children. But, much as I should recommend it to robust children with a solid nervous system, just as emphatically would I forbid it to nervous persons with inherited diabetic tendency for the fear of a possible accident. As I have pointed out previously,[2] the slightest accident that occurs to such persons may cause a traumatic diabetes.

As I have found[3] that alkaline mineral waters are able to improve the assimilative power for carbo-hydrates in cases of alimentary glycosuria, I should consider them as a useful preventive, or at least dilatory agent, against diabetes. They will certainly be more efficacious in such cases than in those in which diabetes has established itself long since in all its vehemence.

If diabetes is still at present an incurable disease—for although the sugar may disappear it nearly always comes back

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[1] "Sur les rapports du diabète avec l'acromégalie et la maladie de Basedow." Communication au Congrès International de Madrid, 25 Avril, 1903.

[2] Lorand : *Die rationelle Behandlung der Zuckerkrankheit*, Berlin, 1903.

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—on the other hand it is by no means impossible to prevent it. It ought to be brought home to the profession that there exists a means of prophylaxis not only against tuberculosis, which at present absorbs all its attention, but also against diabetes. The prophylactic measures against diabetes have also the advantage that they are more easily carried out, and also more efficacious, than those against tuberculosis. It is sad to think how many precious lives could have been saved from early death if many of us could have been sooner aware of this fact.

THE CAUSATION OF CANCER.

By ALEX. THEODORE BRAND, M.D., C.M., Driffield,
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THERE is no subject of more engrossing interest at the present time to the pathologist, the clinician, and the public generally than the problem involved in the elucidation of cancer. The crux of the whole matter is the discovery of the *causa causans*. That there are many contributory causes is very evident, but there is, and can be, only one ultimate cause.

In considering this cause there is a postulate which must be unhesitatingly admitted, viz., that any alleged cause to be acceptable must be absolutely of universal applicability.

Of all the theories hitherto advanced and causes suggested —and they are legion—only one fulfils this postulate, but it does so completely. This one is the theory of the external origin of cancer, *i.e.*, that cancer is an infectious disease.

That the belief in the infectious nature of cancer is rapidly becoming widely held, not only by medical men, but also by the public generally, is undoubted; so much so indeed that the great question now is, "Is cancer infectious or is it not?"

Thus we have two opposed factions, one consisting of those who are out-and-out believers in, and advocates of, infection, of whom I am one; and the other of those who equally strenuously dissent from this belief. There is, no doubt, another class, which however may be disregarded as a negligible quantity, typified by that dilettante agnostic, the man with the "open mind," who knows nothing, who believes that nothing is known, of the causation of cancer, and whose Gallio-like attitude is well calculated to delay indefinitely all progress in its elucidation.

The advocate of infection believes that he has strong grounds for his faith and that he can uphold it by clinical and logical proof. The opponent of infection, on the other hand, simply denies that cancer has an external origin. He does not, and cannot, suggest any other satisfactory cause, for not one other single suggested cause fulfils the inexorable postulate—that of universal applicability. He confines himself to stating objections, some of which are pure fallacies, while others are too frivolous to be seriously considered.[1]

There are, however, objections advanced which are worthy of serious consideration and, because they are founded upon a want of appreciation of the facts of the case, it is the purport of this paper to discuss them.

1. "If there is one and the same parasite at work in all cases of carcinoma, how is it that the secondary growths in each variety so exactly (as a rule) imitate the character of their parent tumours? It seems necessary to suppose that actual fragments of the growth are carried away and develop on their own account."

There is no necessity to *suppose* anything of the sort. It is an undoubted and demonstrated *fact* that actual migration of malignant cells does occur; it is also a fact that the secondary growths *do not* imitate their parent tumours. These secondary growths are simply offshoots from the parent tumour which take on autonomous growth, exactly as a slip from a rose will do. The slip grows into a rose (not, for example, into a geranium), the same as its parent. It does not imitate it, it is actually and really a part of it. This actual migration of malignant cells is generally considered to be extraordinary and without parallel; but when the cause and structure of cancerous metastases are clearly understood, it becomes evident that this migration is not only not at all wonderful, but that it would be extraordinary if such migration did not occur.

Moreover, it is not malignant cells only which migrate. It seems to be overlooked that normal cells also migrate. Without taking into account the leucocyte which finds its way everywhere, we know that bone-cells, for example, also migrate, as evidenced by the inter-fibrillar osseous deposit in muscle, called "myositis ossificans"—a term, by the way, not only absolutely non-descriptive, but also pathologically entirely erroneous.

With regard to malignant-growths, there are two great classes, determined by their source of origin, viz., those derived from the epiblast and hypoblast, called epitheliomata, and those arising from the mesoblast, denominated sarcomata. In both classes the structure consists of cells and stroma, but the relationship of these to each other differs considerably.

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[1] *See* "The Exogenesis of Cancer," *Quarterly Medical Journal*, May 1903.

In the *epitheliomata* the cells are derived from epithelium and lie more or less loosely in the stroma which consists of alveoli formed of connective tissue. The cells are not attached in any way to the alveoli or each other, nor does connective tissue pass between individual cells.

Epithelial tumours are particularly rich in lymphatics. Lymph-channels communicate freely with the alveoli; in fact, the alveoli are really dilated lymph-spaces and form the radicles of the lymphatic trunks which lead to the lymphatic glands. Thus the epithelial cells are bathed in lymph, and being dislodged by the pressure of their own overgrowth, or in any other way, they must of necessity enter the lymph-stream. They are then carried by the current, and are arrested in the nearest lymph-glands.

Blood-vessels permeate the stroma and are often dilated. As they are penetrated by the actively proliferating cells, the latter enter the blood-stream also and are carried far away from the parent tumour.

In the *sarcomata* the arrangement is very different. The cells here are derived from connective tissue, and are embedded in the stroma which also passes between individual cells, and to it the cells are closely attached. There are no lymphatics in sarcomatous tumours, but the smaller blood-vessels are in direct contact with the cells, their walls being often formed of nothing but the cells of the sarcoma, a single layer of endothelial cells separating the blood itself from the cells of the tumour. Thus it is easy, and even inevitable, for the proliferating cells to enter the blood-stream and so be carried to distant parts.

This anatomical arrangement explains the fact that malignant epithelial cells so extensively infect the lymphatic glands and are more rarely conveyed by the blood; while the sarcomatous cells follow the course of the blood-stream, leaving the lymphatic glands unaffected.

These living, infection-laden, autonomous cells, carried away by the lymph- and blood-streams as emboli, on becoming impacted, establish new malignant colonies by the active proliferation of the cells, and the action of the contained infective agent. These colonies, later on, repeat the process, until at length the organism is overwhelmed by their malignant action and the induced constitutional intoxication.

This propagation of cancer by metastasis shows how urgently imperative it is that, so long as removal by the surgeon is our only hope, extirpation of the growth should be carried out at the earliest possible moment before the local disease has had time to become more general and constitutional.

From the foregoing it must be evident that there can be no question of imitation or mimicry, and that genuine migration of the cells of the primary tumour does indeed take place, with formation of metastatic tumours, identical with the parent, as the result. The fact that cancer is spread in this way in the individual must simply be accepted. The fact such extension is not analogous to the method of spread of infection in other diseases may be interesting, but it is otherwise of no moment. It would be absurd to suppose that pathogenesis can be fettered by analogy. One might with equal justice say that cancer cannot be a disease at all, because it is not analogous to any other known disease.

2. A parasitic causation of cancer is improbable, because of the multitude of different growths.

There is, indeed, a great variety of malignant tumours, but they have all the same clinical characteristics. They all infect their environment, tend to ulcerate, extend by metastasis and ultimately destroy the organism.

Pathologically their variety depends solely and entirely upon the tissue of origin and their anatomical structure. As I have already said, there are two great classes of malignant growths, viz., those derived from epithelium and those arising from connective tissue, and all tumours conform in structure to one or other of these classes. The great variety of tumours is due to the great variety of the normal structures which arise from these two classes of tissue, and from which the tumours arise. The structure of these tumours is always the same—cells and stroma—and the variety in form and in consistence depends upon the relative proportion of these to each other, and their environment.

In soft tumours cells predominate, in hard stroma. There is no such entity as a specific cell indicative of cancer, every cell of a malignant growth being an infected cell derived from a normal structure. There is therefore no reason for supposing that a different infective parasite is necessary for each different variety of malignant growth. Electricity, for example, supplies the motive power to a great variety of machinery, but it is not therefore necessary to suppose that it assumes a different form according to the variety of machine driven by it.

3. Distinction between benign and malignant tumours.

Clinically this distinction is exact, and such is freely admitted. Pathologically the distinction is not so clear, because the infective agent cannot be isolated and until this is effected pathologists must rely upon clinical indications.

The pathological distinction is undoubtedly interesting in the abstract, but, after all, it is the concrete, clinical distinction that concerns the unfortunate tumour-bearing individual. The pathologist asks, "Where does a benign fibroma cease and a sarcoma begin?" The clinician replies, "When the benign growth becomes infected from without, the benign tumour becomes malignant."

Benign growths being, for the most part, simple local hypertrophies of normal structures are as liable to infection, after being rendered vulnerable by irritation, injury, or degeneration, &c., as are the tissues from which they spring, and even more so from the fact that their structure is often more hasty and ill-governed, and therefore more liable to external influences, than their parent tissues. No benign tumour forms a metastasis. When one is formed the tumour has ceased to be benign and has become malignant.

One of the most interesting and important points in cancer is the influence of age. Why should malignancy occur so generally in middle and later life? The reason is not far to seek. In early life repair is much in excess of decay, so much so that growth occurs. Later on decay and repair become evenly balanced, and growth ceases, but in middle and old age decay assumes the upper hand, and degeneration occurs; less resistance is offered by the tissues to external stimuli, and so vulnerability is established. Age is therefore a powerful factor in inducing the necessary condition precedent to cancer.

The opponent of the infectivity of cancer may very well say, "You reply to my objections, and no doubt consider that you have answered them satisfactorily, but what are the grounds for your own belief?"

Belief in infection is founded on clinical facts and logical proof. Pathological proof is certainly wanting, but that it will be eventually forthcoming is assured. It is only a question of time. This proof is, however, fortunately, not indispensable, for in many admittedly infectious diseases the infective agent is still only hypothetical.

No one can deny that cancer is most intensely infectious to the individual, and it cannot be gainsaid that all diseases which are infectious to the individual are capable of communication to others, and are also of external origin, therefore cancer is, and must be, of an infectious nature. Infection, undoubtedly differs widely in degree, and fortunately cancer, like tubercle and leprosy, is of a low degree of infectivity, but all the same it *is* infective. The clinical facts are many and convincing—too many to be enumerated here.

The statistics of cancer-districts and houses, and the extraordinary testimony of Dr. Behla regarding the town of Luckau in Bavaria cannot be explained otherwise than by infection. Dr. Behla also supplies a number of cases of *cancer-à-deux*, of which I shall quote but one, viz., that of a man who presented himself at Tross's clinic suffering from malignant disease of the glans penis. On the removal of the growth and its microscopical examination it was found to consist of tissue identical in structure with uterine cervical epithelium, and it was further discovered that the man's wife was suffering from advanced cancer of the cervix uteri. This case alone is sufficient to prove that transference of cancer-cells occurred, that cancer is therefore contagious, and that it is of external origin. The long arm of coincidence cannot be successfully invoked here, nor was this an isolated case, for over sixty such cases have been reported.

Then, again, cancer has been successfully transferred from man to man, and man to lower animals, experimentally, and although such successful experiments are not few in number, even one successful case is sufficient to prove the contagiousness of the disease.

Are we justified, therefore, in sitting still with folded hands and an "open mind" while this vile disease continues to increase by infection? Pending the universal admission of its infectivity should it not, at least provisionally, be considered infectious and action taken accordingly? Prophylaxis, by the thorough disinfection of all rooms and houses occupied by cases of cancer, and the complete destruction by fire of all dressings and dejecta (and such would be cleanly, if nothing else) could not fail to be productive of the greatest good.

It is hopeless to expect the adoption of notification, even voluntary, but medical officers of health might greatly help by instructing registrars to inform them at once of all deaths registered as due to malignant disease, and the coöperation of these medical officers of health with the practitioners signing the death-certificates would afford the best opportunity of thoroughly investigating each case.

What appears to me most deplorable is the utter indifference and supineness of the State with regard to the two great scourges of humanity. Tubercle has been proved to the entire satisfaction of all to be infectious, and yet the Local Government Board has not made the slightest effort to deal with tubercular disease in man (it has in the lower animals!), not even suggesting notification; and it has been left to private enterprise to cope with the evil. So also with cancer.

The investigation of this disease is of national and imperial importance, but no help is forthcoming from the State. Money is lavishly squandered on the army and navy (*vide* the perpetual useless alteration in uniforms), and millions are freely wasted in war, but not one farthing has been offered to further the elucidation of the worst and most hopelessly fatal disease in existence. The only encouragement, even, which has been officially bestowed has been a friendly pat on the back from the Prime Minister who recently offered his best wishes for success to the Cancer Research Committee.

In this country, science owes all its advance to the labours of private individuals, and with regard to the elucidation of cancer we shall have to trust to the coöperation of the clinician and the experimental pathologist.

AN ENQUIRY INTO THE RELATIONS OF LEUCHÆMIA AND TUBERCULOSIS.*

By W. J. SUSMANN, M.A., M.B., B.C. (Cantab.), M.R.C.S., L.R.C.P.

THE fact that within the last few years several cases of leuchæmia associated with tuberculosis have been reported in medical literature, has only served to emphasise the extreme rarity of this occurrence. This rarity is the more striking when we consider that tuberculosis is one of the commonest terminal complications found in numerous wasting disorders, such as diabetes or cirrhosis of the liver, whilst in Hodgkin's disease, which has a somewhat close superficial resemblance to leuchæmia, it is so frequently found (Wactzoldt and Askanazy,[1] C. H. Lewis,[2] Michell Clarke[3]), that some authorities are of the opinion that Hodgkin's disease is primarily a form of tubercular infection. In most of the recognised text-books in the English language there is no mention at all of the association of leuchæmia with tuberculosis.

I have been able to collect from the literature in all 25 cases of leuchæmia associated with tuberculosis. Of these 25 cases, 10 are mentioned by Hirschfeld and Tobias[4] in their paper of February, 1902. It must be here pointed out that in deaths from leuchæmia the post-mortem appearances are frequently those of an acute tuberculosis, and it is necessary to be on one's guard in diagnosing the presence of tubercle from the macroscopic appearance alone without the aid of the microscope. Gowers,[5] in his monograph on Leuchæmia, has been careful to point this out. He says that "phthisis is rare, but the appearances in the lungs in one or two cases have been those of tubercle, and, in a few cases, tubercles have been found. On the other hand, extravasations of blood into the lungs are common, and they may undergo caseation and so closely simulate a tubercular process. Cavities may even result from lymphoid-infiltration." I have tried to eliminate this source of error as far as possible by only accepting those cases which are recorded by competent observers, or in which the diagnosis has been confirmed, macroscopically and microscopically, by the post-mortem examination.

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* Abstract of a thesis for the degree of M.B. at the University of Cambridge.

With a view to testing the rarity of the combination I have examined the post-mortem records of St. George's, Westminster, Middlesex, St. Thomas's and St. Bartholomew's Hospitals from January 1890 to December 1901. During this period of 11 years, out of a very large number of post-mortem examinations, there were in all 41 necropsies on cases of leuchæmia. In only one of these (No. 25 of my series) was there good evidence of the presence of tuberculosis. These figures are small, but the cases of leuchæmia treated in hospital practice bear a very small proportion to other hospital cases.

G. Dock,[6] in his Philadelphia practice, had a particularly high percentage, ·113, but, taking an average from a large number of cases treated in Vienna, Berlin, Prague, Rostock, Stockholm, and Philadelphia, the percentage works out at ·067. From these figures it will be seen that it is not an easy matter to obtain large statistics.

Basing our calculations on these 41 necropsies, we may say that in every hundred post-mortem examinations we should expect to find 2·4 deaths from leuchæmia complicated with tuberculosis. From statistics, based on the Registrar-General's reports, it is usually stated that one person in seven dies from tuberculosis. A number of errors creep into these reports, and these figures are probably too high. Latham[7] states that "at least one in ten dies from tuberculosis in this country."

In 7,200 necropsies of all diseases which I examined, there were 827 deaths from tuberculosis or complicated with tuberculosis, *i.e.*, in every 100 deaths 11—12 were due to or complicated with tuberculosis. In every 100 deaths from leuchæmia we might therefore expect from these figures to find at least 10 deaths complicated with tubercle. Instead of this we find only 2·4 deaths. There is therefore a very marked antagonism between these two diseases.

In some of the cases which I have collected from medical literature the details to be obtained are extremely meagre, but I have consulted the actual record of the writer wherever this has been possible. In the appendix will be found, in tabular form, some of the results obtained from an analysis of these 25 cases. This analysis gives us the following results:—

Sex.—Of 21 cases in which the sex of the patient is mentioned 18 were males and only 3 females. Although the number of cases is not very large this result is remarkable. That leuchæmia affects the male sex more frequently than the female is well known. Birsch-Hirschfeld, quoted by Osler,[8] found that, in 200 cases, 135 were males and 65 females, *i.e.*, a proportion of 2 to 1. Between the years of 18 and 50 tuberculosis does not affect one sex more than the other. Percy Kidd,[9] quoting Dr. W. Ogle's figures, states that, "age put aside, sex makes no difference in the mortality from tuberculosis. Between 5 and 35 the mortality amongst females is in marked excess, whilst from 35 onwards the male mortality becomes gradually higher." When the two diseases, leuchæmia and tuberculosis, are combined, this disproportion between the male and female mortality is markedly increased. Six males are now affected for one female, whereas the proportion before was two to one.

Age.—With regard to age, 17 cases were between the years of 18 and 50, 2 cases were under 18, and 2 cases over 50. The youngest was 9 years of age, the oldest was 58.

Type of Leuchæmia.—In 5 cases the leuchæmia was definitely, whilst in 2 (Case 1 and Case 19) it was probably, of the spleno-medullary type. In 17 cases the leuchæmia was of the lymphatic type. The lymphatic type was, therefore, two and a half times as common as the spleno-medullary type. This again is contrary to what is found in uncomplicated cases of leuchæmia. Osler states that the spleno-medullary type is the commonest, whilst the lymphatic is rare. Of 24 cases which came under his care, 15 were spleno-medullary and 9 lymphatic. The rarity of the combination of spleno-medullary leuchæmia and tuberculosis was first pointed out by Elsner and Groat.[10] Since the publication of their case in 1901, four more cases have been recorded within the last three years. It is possible, therefore, that as the result of more careful and accurate examinations of the blood this disproportion may be modified.

Precedence of Tuberculosis or Leuchæmia.—This subject would appear to divide itself into three varieties:—

Class A.—The tubercle is old and obsolete; the advent of the leuchæmia does not affect it.

Class B.—The tubercle is old and latent, and the advent of the leuchæmia sets it aflame again.

Class C.—The tubercular infection occurs during the progress of the leuchæmic disorder, owing to the lessened resistance of the individual.

Although dogmatic statements cannot be made in every case, it would appear that 16 cases from my series can be placed in Class B.; 5 cases (Nos. 1, 6, 16, 20, and 25) in Class C.; whilst 2 (Nos. 11 and 22) come in Class A. The following notes and a reference to the appendix will show the evidence from which these figures have been obtained:—

Case 9.—There was no clinical evidence during life to show the presence of tubercle, and the caseating condition of the glands, found *post mortem*, does not present the appearance of a recent tubercular process.

Case 12.—The swelling of the glands in early life points to an early tubercular infection.

Case 13.—The tubercular symptoms—the cough, &c.—preceded the increase in size of the spleen.

Case 14.—There is a history of tuberculous glands at 10 years of age in a man of 26. The subsequent sudden swelling up of the glands is supposed by Lichtheim to be the result of the leuchæmia lighting up the old tubercle.

Case 18.—Junger thinks that the tubercle was latent. Then, after the leuchæmic infection had been in process three months (? originated from an abscess under the tongue), there was an awakening of the tubercular process. This resulted in a general infection of the lymphatics—the seat of the leuchæmic process being less resistant to tuberculosis than the normal ones.

Case 23.—Hirschfeld and Tobias think that the evidence of the old tubercle at the apices of the lungs points to the fact that the advent of the leuchæmic condition lighted up again the tubercle.

In Cases 1, 6, 16, 20, 25 it is presumed that the tubercular mischief supervened on the leuchæmia for the following reasons:—There was no clinical history of tuberculosis until a very late date; the course of the disease was extremely rapid upon the advent of the tubercular symptoms; and the post-mortem evidence showed only recent tubercles and no signs of any old tubercular mischief. In Cases 11 and 22 an old tubercular cavity was found accidentally *post mortem*. It would, therefore, appear that the conditions described under Class B. are most commonly met with.

Blood-changes found in combined Leuchæmia and Tuberculosis.—With regard to the changes in the blood, the most noticeable point is that as the tubercular process advances the leuchæmic character of the blood tends to be modified or even lost. The point most often noted is the diminution in the number of leucocytes. This is well shown in nearly every case. This phenomenon is not of course peculiar to the association of leuchæmia and tuberculosis.

Fraenkel[11] in 1895, Marischler[12] in 1896, and Weil[13] in 1900 have made exhaustive collective studies of the complications of leuchæmia, and have quoted cases in which erysipelas, cellulitis, angina, pneumonia, typhus, &c. have supervened. The fall in the leucocytosis with the advent of the second disease is, according to Weil, extremely marked. In one case reported by Müller,[14] where the leuchæmia was complicated by septicæmia, there was an increase in the percentage of polymorphonuclear cells, *i.e.*, there was a general leucocytosis. In another case of leuchæmia complicated by acute rheumatic arthritis reported by Richter,[15] the condition of the blood remained unaltered. In my series of cases the fall in the leucocytosis was particularly well-marked in Cases 14, 18, 19, 20, and 25.

With regard to the changes in the characters of the leucocytes themselves, the observations are not very conclusive. Whilst Lichtheim (Case 14—lymphatic) and Sturmdorf (Case 21—spleno-medullary) found that with the reduction of the leucocytes there was also a complete disappearance of the characteristic features of the leuchæmic blood, Elsner and Groat (Case 20), Hirschfeld and Tobias (Case 23), and Murrell (Case 25) found that although there was a steady decrease in the total number of leucocytes, the blood retained the characteristic features of spleno-medullary leuchæmia, as shown by the presence of the myelocytes, although in reduced numbers. The only exception to the reduction in the number of leucocytes was in G. Parker's case (No. 24), where the number of myelocytes is said to have increased just before death.

Sturmdorf, in connection with Case 21, points out that the occurrence of a febrile temperature was characterised by a change in the relative proportion of the myelocytes and polymorpho-nuclear cells. This is of course only what one would expect.

Weil, after studying the effect of numerous intercurrent affections on leuchæmia, came to the following conclusion:—"In spleno-medullary leuchæmia, as the result of an intercurrent affection, there is a diminution in the number of leucocytes, and a disappearance, more or less, of the mono-nuclear cells, which are replaced by the polymorpho-nuclear cells." This is exactly the condition found by Sturmdorf (Case 21).

Quincke, Stintzing, Lichtheim, Baldwin, and Murrell all noted a marked diminution in the size of the spleen and glands as the tuberculosis advanced. Quincke states that, as the phthisis advanced, the leuchæmic condition improved; whilst Stintzing declares that the phthisis was gradually arrested and the leuchæmia also steadily improved.

In this connection it is interesting to note that injections of tuberculin have been used by Heuck[16] and Beitzke[17] as curative measures. Heuck used them in one case and Beitzke in six. After each injection there was a fall in the number of leucocytes, but not to a very marked extent. Two of Beitzke's results were vitiated by the simultaneous use of arsenic, and the results taken altogether are inconclusive; but there seems to be some scope for further experimentation in this direction.

Dr. Rolleston has used anti-streptococcic serum in one instance. After each injection there was a fall in the number of leucocytes, but sufficient time has not yet elapsed in order to judge whether the result will be permanent or not.

Extracts of spleen have been used by Jacob,[18] whilst Richter,[15] quoted by Fraenkel, used spermin.

Effect on the Duration of the Leuchæmia.—The question as to whether the duration of the leuchæmic disorder is prolonged or shortened by the association with tuberculosis, is even more difficult to determine. Exact dates as to the onset of either one disease or the other are impossible to obtain. On the whole, looking at the post-mortem records, it would appear that, whilst the tubercular infection up to a certain point exercises an antagonistic influence on the leuchæmia, the resistance of the individual, and more particularly his lymphatic system, is so undermined by the leuchæmic process that there is a tendency for the case to terminate by the supervention of an acute miliary tuberculosis.

That the association of tuberculosis and leuchæmia is rare I think we may consider as proved from the figures quoted. That when in combination they appear to exercise an antagonistic effect, would also appear probable from the fall in the number of leucocytes and the diminution in the size of the spleen and glands. If we are right in these conclusions, the question that naturally arises, is—Why is the leuchæmic patient protected from tuberculosis? I offer the following suggestions, but it is unfortunate that experimental evidence cannot be adduced, for two reasons—first, the extreme rarity of the association of the two diseases, and secondly, the fact that even when present, the secondary infection of tuberculosis may not be discovered or even suspected until the post-mortem examination is made. This latter was the case in the only instance of the associated diseases which I have personally seen (Case 25).

Contrasting the two diseases, leuchæmia and tuberculosis, the most striking difference, putting aside the presence of the tubercle-bacillus itself, is the presence of a marked mononuclear leucocytosis in leuchæmia as against the absence of a leucocytosis in tuberculosis. I would suggest that this monoleucocytosis acts in a manner antagonistic to tuberculosis in the following way:—

(1) By increasing the quantity of nucleo-albumin present in the body.

(2) By increasing the phagocytic action of the blood.

Buchner[19] found that leucocytes gave rise to certain substances rich in nucleo-albumin to which he gave the name of alexines. These alexines, or the serums containing them, have marked bactericidal powers. Any increase in the quantity of nucleo-albumin present in the body will tend, therefore, to act as a protective agent against the invasion of bacteria.

Are the nucleo-albumins present in excess in leuchæmic patients? We have good reason for believing that they are since it is a well-recognised fact that uric acid is present in excess in leuchæmic patients (Kühnau[20]). J. Horbaczewski[21] and Kühnau have both demonstrated the fact that uric acid can be produced from the white blood-corpuscles. The derivatives of uric acid are, in fact, urea and nuclein, and these latter products are derived from the ingestion of nitrogenous food or from nucleo-albumins. That there is an excess of nucleo-albumin produced in the tissues of leuchæmic patients we may, therefore, consider proved.

But, if an excess of nucleo-albumin acts as a protective agent against tubercle, in tuberculous patients nucleo-albumin should be present in diminished quantities. This is actually the fact. The chlorides, phosphates and acids are markedly deficient in the tissues of tuberculous patients. The production of urea and kreatinin is also diminished in tuberculosis (Berheim). H. Harper,[22] contrasting the diminished output of nitrogenous products in tuberculous patients and the excessive excretion of urea and uric acid in gouty patients, and recognising an antagonistic influence of the gouty upon the tubercular habit (Sir Dyce Duckworth), came to the conclusion that "nitrogen and nitrogenous products are the remedies *par excellence* for the tubercle bacillus." He prescribes "urea" as the most suitable means of increasing the amount of nitrogenous intake. Latham (*loc. supra cit.*), whilst agreeing that it is advisable to increase as far as possible the nitrogenous intake, is not convinced of the merits of urea. Berheim, on somewhat similar reasoning, has increased the acidity of the patient by administering phosphate of creosote or phosphoric acid.

De Backer introduced the ferment-treatment, of which Manders[23] has given a full account. The ferment is administered as pure yeast. It is found that pure yeast-cells are distinguished by the large amount of phosphorus which they contain, thereby closely resembling the leucocytes and blood-plasma in their chemical constituents. The nucleins, obtained from yeast, also differ very little from those of animal cells. Whilst by these remedies we attempt artificially to increase the amount of nucleo-albumin present in the tissues of the tuberculous patient, in the leuchæmic patient, as the result of the pathological leucocytosis, this condition is already actually present, and therefore the leuchæmic soil may reasonably be supposed to exert an antagonistic effect upon the tubercle-bacillus. Manders claims for the yeast-cells an additional property—that of englobing the tubercle-bacillus (p. 178). That vegetable cells have any phagocytic action within the body is not as yet, however, generally recognised. That the leucocytes of the body are phagocytes is well known. According to Manders, leucocytes have varying powers of bactericidal action. He states that the myelocytes, although slow in action, are more powerful phagocytes than the mononuclear cells, whilst these in turn are more powerful than the polymorpho-nuclears (page 156). If this is so, the association of tuberculosis and the spleno-medullary type of leuchæmia (the chief characteristic of which is the presence of myelocytes) ought to be the rarest combination. That this is so we have already seen.

CONCLUSIONS.

It would appear then that :—

(1) Tuberculosis is rarely found in combination with either the spleno-medullary or lymphatic form of leuchæmia.

(2) When the combination does occur, it is two-and-a-half times as common in the lymphatic as in the spleno-medullary form.

(3) It is six times as common in the male as in the female.

(4) The tuberculosis may be found latent and obsolete.

(5) The tuberculosis may supervene as a terminal infection.

(6) The tuberculosis may be latent and be lighted up as the result of the leuchæmic infection.

(7) This last is the condition most commonly found.

(8) When the two diseases are combined, there is a tendency for the total number of leucocytes to be diminished, and the spleen and glands to decrease in size.

(9) This antagonism is possibly due to the excess of nucleo-albumin and the increased phagocytic power of the blood, both of which conditions are present in the leuchæmic patient.

APPENDIX OF CASES.

Case.	Reference.	Sex and Age.	Occupation.	Type of Leuchæmia.	Relation of Tubercular Symptoms to Leuchæmic Symptoms.	Evidence of Tubercle.	Approximate Date of		Date of Death.
							Onset of Tubercle.	Onset of Leuchæmia.	
1	*Virchow's Archives.* Bd. II., p. 587.	M. 38	Cabinet-maker.	? Myelogenous.	Tubercle supervened on leuchæmia.	P.M.: Tubercles in lungs and pleuras.	Oct. 1848	1844	Nov. 2, 1848.
2	Bamberger. *Schmidt's Jahrbücher*, 1858. Bd. XCVII., p. 204.	M. 35	Brewer	Lymphatic	Signs of old tubercle -	P.M.: Caseous tubercle and fibrous patches in lungs. Grey tubercles on pleuras.	?	1854	Oct. 1855.
3	Hounsell. *Schmidt's Jahrbücher.* Bd. CXXII., p. 47, or *Med. Times and Gazette*, 1863, Dec. 12.	M. 18½	—	? Lymphatic	Tubercle probably preceded leuchæmia.	P.M.: Tubercle of both lungs and meninges.	?	June 1863	? Nov. 1863.
4	Ollivier et Ranvier. *Arch. de Physiologie*, 1869, p. 416.	M. 40	—	Lymphatic	Tubercle preceded leuchæmia.	P.M.: Old tubercle at both apices Miliary tuberculosis of peritoneum.	?	Jan. 1, 1866	Mar. 11, 1868.
5	Robin. *Soc. Méd. des Hôpitaux*, 1883.	M. —	—	—	Tubercle was old - -	P.M. - - - - -	?	—	—
6	Quincke. *Tageblatt der Natürforscher und Aerzte in Heidelberg*, 1889, p. 405.	M. 38	Labourer	Lymphatic	Acute miliary tuberculosis supervened on leuchæmia. Reduction in leucocytosis.	P.M. - - - - -	? June 2, 1886.	? Mar. 1886	June 5, 1865.
7	Stintzing, *Tageblatt der Natürforscher und Aerzte in Heidelberg*, 1889, pp. 405, 405.	—	—	Lymphatic	Tubercle was old and chronic. As it progressed the leuchæmia improved.	Tubercle-bacilli in sputum.	—	—	—
8	Heuck *Deutsche Med. Wochenschrift*, 1891, p. 748.	—	—	? Lymphatic	—	P.M. - - - - -	—	—	—
9	Francksen. *Inaugural Diss., Göttingen*, 1892.	M. 53	—	Lymphatic	? Probably tuberculosis preceded leuchæmia.	P.M.: Caseating tuberculosis of glands, spleen, and liver.	? 1891	July 7, 1891	Nov. 8, 1891.

Case.	Reference.	Sex and Age.	Occupation.	Type of Leuchæmia.	Relation of Tubercular Symptoms to Leuchæmic Symptoms.	Evidence of Tubercle.	Approximate Date of		Date of Death.
							Onset of Tubercle.	Onset of Leuchæmia.	
10	De Roth. *Inaugural Diss., Geneva*, 1895.	—	—	? Lymphatic	——	P.M.	—	—	—
11	Musser and Sailor. *Transactions of Assoc. of American Physicians*, 1896, Vol. XI.	M. 31	Labourer	Lymphatic	Tubercle old .	P.M.: Tubercle at apex of right lung.	?	Mar. 1895	Feb. 1896.
12	M. Volpe. *Arch. internaz. di Med. e Chir.*, Napoli, 1896, pp. 161–175.	M. $9\frac{7}{12}$	—	? Lymphatic	Tubercle probably preceded leuchæmia.	No P.M. Cultures from spleen produced tuberculosis in rabbits.	No date, 2 years of age.	No date, æt. 4.	Æt. 9 years and 7 months.
13	Volpe. *Ibid.* . .	M. 19	Shoemaker	? Lymphatic	Tubercle probably preceded leuchæmia.	Tubercle-bacilli found in sputum. P.M.: Cavities in lungs.	No date, æt. 17.	No date, æt. 19.	Æt. 19 years and 3 months.
14	Lichtheim. *Deutsche Med. Woch.* Vereinsbeilage, 1897 Sept. 30th.	M. 26	—	Lymphatic	Tubercle preceded leuchæmia. As tuberculosis advanced, leucocytes diminished in number (250,000 to 8,900 per cmm.).	P.M.: Caseous tuberculosis of glands of neck. Early tuberculosis of bronchial glands. Tubercular pleurisy, pericarditis, peritonitis.	Æt. 10	Æt. 24	Æt. 26.
15	Baldwin. *American Journ. of Med. Sci.*, 1899. June, p. 656.	M. 47	Machinist	Lymphatic	Tubercle preceded leuchæmia. Leucocytosis progressive: tuberculosis stationary.	P.M.: Old chronic tubercle in lungs.	1894	Sept. 1894	Dec. 1897.
16	Beitzke. *Ueber Beeinflussung der Leukämie durch complicirende Krankheiten.* Diss. Kiel, 1899.	M. 48	Farmer	Lymphatic	Acute tuberculosis supervened on leuchæmia. Leucocytosis diminished.	P.M.: Acute miliary tuberculosis of lungs. Tubercular ulcer of vocal cords. Tuberculosis of liver, kidneys, and pleura.	Jan. 1891	Mar. 1891	June 1891.
17	Bruckmann. *Arbeit aus dem Pathol. Institut in Tübingen.* Bd. ii. 1899. Heft 3.	M. 13	—	Lymphatic	Tubercle of glands old and probably primary.	P.M.: Old tubercle of glands. Recent miliary tuberculosis of diaphragm.	—	Jan. 1892.	June 1892.

18	Junger. *Virchow's Archives*, CLXII., 1900, p. 283.	M. 25	Labourer	Lymphatic	Tubercle probably preceded leuchæmia. Leucocytosis fluctuated.	P.M.: Miliary tuberculosis of peritoneum. Glands showed tuberculosis.	Nov. 1899	Dec. 1899	Mar. 1900.
19	Bezançon et Emile Weil. *Soc. Méd. des Hôpitaux*, 1900, p. 804.	F. 44	—	Spleno-medullary.	Tubercle old and latent. Recent tuberculosis in lungs. Leucocytosis diminished.	P.M.: Tubercles in lungs -	?	Dec. 1899	Jan. 1900.
20	Elsner and Groat. *Amer. Journ. of Med. Sci.*, 1901, Mar.	M. 40	Labourer	Spleno-medullary.	Tuberculosis probably supervened on leuchæmia. Leucocytosis diminished.	Tuberculous bacteria found in sputum.	Nov. 1899	April 1899	—
21	Sturmdorf. *Amer. Journ. of Med. Sci.*, 1901, Aug.	F. 25	—	Spleno-medullary.	Tubercle probably preceded leuchæmia. Leucocytosis nearly stationary. Polymorphs increased with rise of temperature, myelocytes diminished.	—	? Apr. 1901	?	—
22	Saundby. *Brit. Med. Journ.*, 1901, p. 1.	M. 58	Engraver	Lymphatic	Tuberculosis old and not advancing. Slight diminution in leucocytosis.	P.M.: Chronic fibroid and caseating tubercle at apex of right lung.	?	Feb. 26, 1900.	Mar. 4, 1900.
23	Hirschfeld and Tobias, *Deutsche Med. Wochen.*, 1902, Feb. 6.	M. 31	—	Spleno-medullary.	Tubercle probably preceded leuchæmia. Leucocytosis fluctuated.	P.M.: Indurated pigmentation and old tubercle of both apices. Miliary tuberculosis of lungs, pleuræ, and liver.	?	Jan. 1899.	Sept. 1900.
24	Parker. *Brit. Med. Journ.*, 1902, May 10.	M. 34	Engine-fitter.	Spleno-medullary.	Tubercle preceded leuchæmia. Leucocytosis fluctuating.	No P.M. Tubercle-bacilli in sputum.	1893	May 1899	Feb. 15, 1902.
25	Murrell. *Lancet*, 1902, July 19.	F. 21	—	Spleno-medullary.	No def. evidence. Probably tuberculosis supervened on leuchæmia. Leucocytosis diminished.	P.M.: Tuberculosis in lungs. ? Early tuberculosis in liver.	?	Sept. 1900	May 1902.

REFERENCES TO LITERATURE.

[1] Wactzoldt and Askanazy; Ziegler's *Beiträge*, Bd. III.
[2] Lewis, C. H.: *Med. Record*, N.Y., 1897, August 28.
[3] Micheil Clarke: *Brit. Med. Journ.*, 1901, Vol. II., p. 701.
[4] Hirschfeld and Tobias: *Deutsche Med. Wochensch.*, 1902, February 6.
[5] Gowers: Reynold's *System of Med.*, Vol. V.
[6] Dock, G.: *Phil. Med. Journ.*, 1900, March 31.
[7] Latham: *Pulmonary Consumption.*
[8] Osler: *Princip. and Prac. of Med.*, 1901.
[9] Kidd, P.: Clifford Allbutt's *Syst. of Med.*, Art., "Pulm. Phthisis."
[10] Elsner and Groat: *Amer. Journ. of Med. Sci.*, 1901, March.
[11] Fraenkel: *Deutsche Med. Wochensch.*, 1895, pp. 35-45.
[12] Marischler: *Wien. Klin. Wochensch.*, 1896, p. 686.
[13] Weil: *Gaz. Hebd.*, 1900, No. 70.
[14] Müller: *Wien. Klin. Wochensch.*, 1896, July.
[15] Richter: *Deutsche Med. Wochensch.*, 1895.
[16] Heuck: *Deutsche Med. Wochensch.*, 1891, p. 748.
[17] Beitzke: *Diss. Kiel*, 1899.
[18] Jacob: *Deutsche Med. Wochensch.*, 1894, p. 641.
[19] Buchner: *München. Med. Wochensch.*, 1893, p. 449.
[20] Kühnau: *Zeitschrift für Klin. Medicin*, Berlin, 1897, Bd. XXXII., p. 482.
[21] Horbaczewski, J.: *Wien. Sitzungsbericht*, Bd. XCVIII., Abtheilung III., p. 301.
[22] Harper, H.: *Brit. Med. Journ.*, 1902, Vol. II., 1235.
[23] Manders, H.: *Ferment Treatment of Cancer and Tuberculosis* (Rebman, 1898).

A REVIEW OF RECENT LITERATURE ON DIPHTHERIA.

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DIAGNOSIS.

ALTHOUGH there has been nothing of very great originality on the subject of diphtheria published during the last twelve months, still a considerable number of interesting articles have appeared. The question of diagnosis is discussed very fully by Marfan (*Gazette des Hôpitaux*, Mars 14, 1903). His very interesting paper commences with a discussion on the value of bacteriological diagnosis. It is admitted that a single culture does not give an infallible result. Again, there is the difficulty caused by the pseudo-diphtheria group of bacilli. Grancher, another great French authority, is quoted as going so far as to say that bacteriological diagnosis is neither quicker nor surer than clinical diagnosis. With a view of obtaining light on this vexed question, Marfan has utilised the large amount of material afforded him by the diphtheria-wards of the Enfants Malades of which he has been in charge for two years. Over two thousand cases have been bacteriologically examined, and the results have been compared with the clinical features of the cases. Where any doubt existed, animals have been inoculated, and the results of inoculation have been controlled, when necessary, by the injection of antitoxine into the inoculated animals. The conclusion arrived at is that there is much less discrepancy between clinical and bacteriological results than is sometimes asserted, and that this discrepancy is often due to errors on the part of the physician or bacteriologist.

As a general rule the diagnosis can be made clinically, if not with absolute certainty, at any rate with a probability which amounts to almost the same thing. As to bacteriological diagnosis, while it may not be absolutely infallible, yet if performed with due care and by an expert, it nearly always settles any doubt left after a clinical examination. Except, therefore, in very rare instances, it is easy to decide whether to give antitoxine without waiting for a bacteriological examination.

On the question of clinical diagnosis Marfan discusses at length the appearances of different kinds of sore-throat. He does not appear to believe in a simple catarrhal diphtheria, the "diphtheria without membrane," which is now usually described in the text-books. The arguments adduced for its existence are, he points out, firstly, that in cases of laryngeal diphtheria bacilli are found in the fauces which may be only slightly injected and show no membrane; and, secondly, that every diphtheria commences with an initial erythematous stage before the membrane appears. As regards the first argument he says, very justly, that the bacilli are probably coughed up from the larynx, and that it does not follow that faucial diphtheria exists at all; and, as to the second point, he has not in his experience met with red and injected throats, which ultimately took on an appearance of false membrane, except in the case of scarlatina. In the presence, therefore, of what he calls "*une angine rouge*" he thinks that the physician may safely dismiss the idea of diphtheria from his mind. A more difficult question is that of the combination of diphtheria with scarlatina. Marfan considers that the scarlatinal throats which present a pseudo-membranous appearance are rarely diphtheritic, and that in any case a bacteriological examination is required to make a diagnosis. The result of this may be waited for safely without injecting antitoxine, provided that there is only slight adenitis, that there are no nasal or laryngeal symptoms, and that the patient has not been exposed to diphtheria. In such cases it would be necessary to give the serum at once.

Another interesting point is, how far can true diphtheria assume the appearance of a follicular tonsillitis. Most physicians who have seen much diphtheria will agree with Marfan that certain true cases, especially when the larynx is involved first, do present such an appearance. In his wards he found that 80 per cent. of the cases of follicular tonsillitis accompanied by laryngitis were diphtheritic, whereas under one-half per cent. of follicular cases with no laryngeal symptoms were found to be diphtheria. In a case of doubt it will be always found that the exudation tends to spread over the surface of the tonsil very rapidly, if diphtheritic; and if such a spread does not occur in 24 or 48 hours, a negative diagnosis can be safely made. As long as the condition remains follicular, it is quite unnecessary to give antitoxine, always provided there are no nasal, laryngeal, or marked glandular symptoms, and that an epidemic is not present.

Marfan's general conclusions are briefly as follows. A throat which shows any false membrane must be considered as diphtheritic, and treated as such, unless it occurs in the course of convalescence from scarlatina, or follows a herpes or syphilis of the pharynx, or a tonsillotomy, or a cauterisation of the throat. Even in the latter cases it is wise to make a bacteriological examination. Secondly, if the pseudo-membranous condition complicates scarlatina, syphilis, or herpes; if the condition seems follicular or is ulcerative, he concludes that diphtheria is improbable. The presence, on the other hand, of laryngeal or nasal symptoms should lead to a positive diagnosis, even in very unsuspicious throats. In young children white patching of the throat is usually diphtheritic. In all doubtful cases inject antitoxine.

The article, it will be seen, is nearly above criticism, the one unsatisfactory part about it being the lightness with which the writer seems to regard the probability of a concurrence of scarlatina and diphtheria. Such a coëxistence of diseases is seen only too frequently in our own hospitals, and diphtheria may complicate any stage of scarlatina. On the other hand, it must be frankly admitted that many scarlatinal throats are extremely like those seen in diphtheria, and yet are not often followed by paralysis.

Two other papers of interest refer to questions of diagnosis of diphtheria. Dr. Foord Caiger (*Clinical Journal,* Dec. 31, 1902) has not nearly the same confidence in bacteriological examination as the French observer quoted above. He points out that not only do we come across typical, and even severe, or fatal attacks with clinical appearances distinctive of diphtheria, in which careful and repeated examination of the throat and secretions fails to reveal a single diphtheria-bacillus, but, on the other hand, it is not uncommon to find virulent bacilli in the throat-secretion of perfectly healthy persons. In a series of 140 cases of scarlet fever examined bacteriologically by Professor Sims Woodhead a large proportion were found to contain diphtheria-bacilli. Ranke of Munich, moreover, states that 52 per cent. of his scarlatinal throats contained the bacillus in question. In the rhinorrhœa of scarlet fever the presence of the bacillus is also common. While such persons may be a danger to the public, they are certainly not suffering from diphtheria. On the other hand, a positive bacteriological examination of a throat presenting definite follicular exudation, or from the larynx in a case of croup, even without visible faucial exudation, may be taken as sufficient evidence of diphtheria. In the case of a negative examination of a case in which the clinical features point to diphtheria, it is wiser to ignore the bacteriological evidence altogether.

In connection with such cases as Caiger here mentions, it would be a great advantage if hospital statistics of diphtheria discriminated between those cases in which there is merely a positive bacteriological report and those in which the clinical symptoms are in reality those of diphtheria. Last year I admitted into the Edinburgh City Hospital some 15 or 20 cases from a Children's Hospital, in all of which bacilli were found in the nasal secretion, but none of whom suffered from diphtheria. I could have decreased my death-rate appreciably for the year had I classed these cases as diphtheria; they were all notified as such. It would be interesting to know in view of the very low death-rates of some institutions if such cases are shown as diphtheria in the statistics. While no doubt a fever-hospital may be a very suitable place for them, they are in my opinion certainly not cases of diphtheria, and again, if I isolated every case in the scarlatina-wards in which bacilli was found, half the patients would probably be isolated, which would be manifestly absurd. In my experience they are seldom a danger to others. Indeed, it almost is coming to this, that without inoculation-experiments a bacillus means little or nothing, and inoculation-experiments require time. In conversation with physicians in charge of large numbers of diphtheria-cases I usually find that they diagnose a case clinically, give antitoxine, and do not pay too much attention to the bacteriological report.

But to return to Dr. Caiger's paper: he speaks favourably of the direct cover-glass preparation from the throat-swab. There is no doubt that with a little practice much can be learned by such an examination, as it gives the relative number of various organisms in the throat; and if, as he recommends, Neisser's double stain is used, the identity of the diphtheria-bacillus is practically assured. In reference to this method, we may remark, however, that Wicliffe Peck (*Lancet*, January 10, 1903) considers that it has a very limited value, not being reliable in more than 60 per cent. of swabbings. He has therefore tried staining with Löffler's methylene-blue, 3 to 4 seconds, with Neisser's brown (0·2-per-cent. aqueous solution), 30 seconds, as a counter stain, and has obtained much better and more reliable results.

In very much the same spirit Ball (*Annales de Médecine et Chirurgie Infantiles*, Juin 1, 1902) complains that clinical signs are not studied as attentively as they used to be. There is too great a tendency to throw a hurried glance at the throat, and then to wait for a bacteriological report. On the other hand, it should be remembered that clinical diagnosis is as important as ever, and has also the advantage that it can be made on the spot. In all but very mild cases, when there is any doubt clinically, it is necessary to use antitoxine without waiting for the report. In all cases of croup the serum should be injected at once.

We have seen above that Marfan is inclined to throw doubt upon the existence of a "Simple Catarrhal Diphtheria." This is not the view of Josias and Tollemer (*Annales de Médecine et Chirurgie Infantiles*, 1st November, 1902), who give an account of 709 children admitted in one year to the Bretonneau hospital as suffering from diphtheria. They class their cases as bacteriological and clinical diphtheria respectively. Under the first head are 44 cases in which the clinical appearance was that of a simple catarrhal sore-throat, occasionally purulent, but never membranous, and in which the Klebs-Löffler bacillus was present. Such cases, if not promptly treated, may in their opinion take on a membranous character. In other instances they may depend upon a low degree of virulence on the part of the bacillus or a high resistance on the part of the child. All the same, the writers seem doubtful whether such cases should be called true diphtheria. They account for the large number observed by the fact that the relatives of the clinical cases were bacteriologically examined, and if the bacillus was present, were admitted to hospital. As regards the clinical cases, interesting figures are given showing the death-rate according to the bacteriological examination. Of 178 cases in which the bacillus only was found there were only 7 deaths, a rate of 3·93 per cent. Of 131 cases in which, besides the bacillus, other organisms, streptococci, staphylococci, pneumococci, and colon-bacilli were present, 13, or 9·92 per cent., died. Of 153 children with a laryngeal complication the death-rate was 20·91 per cent. Simple laryngeal diphtheria gave a percentage death-rate of 22, while so-called malignant or hypertoxic diphtheria was responsible for the death of 60 per cent. of those who suffered from it. Of these malignant cases those in which there was an association of organisms proved most fatal. The total death-rate of all cases came to 13·10 per cent.

DIPHTHERIA AS A DISEASE OF PERSON.

Heaven (*Public Health*, June, 1903) has an interesting article on "Diphtheria as a Disease of Person." In his opinion, after the experience of an outbreak at Bristol, diphtheria is a personal disease, personally spread; and to deal with it effectually measures should be adopted to check the personal carriage of infection; other conditions, such as filth-nuisances of any kind, receiving due attention as predisposing causes. In examination of cases or of contacts the nose as well as the throat must receive attention. In the outbreaks with which he had to deal the nose played a prominent part, and it is interesting to note, after what has been said above, that the nasal bacilli were found virulent by Klein. The best methods appear to be removal to hospital of all bacteriological cases, proper disinfection, bacteriological examination of cases and contacts, and treatment of "suspicious" persons. By these last are to be understood those cases in which suspicious bacilli only were found. They can be successfully dealt with either at home or in special buildings. Success depends on carrying out the methods adopted uniformly over the whole district affected, and steadily continuing them till the outbreak has ceased. It is not necessary to close the schools, as it is easier by means of them to keep the children under supervision. Heaven very justly submits that it is hard to see how prophylactic injections can favourably affect persons carrying about infective bacilli in their nose, as regards the probability of their infecting others. He argues that the Hofmann bacillus should be taken seriously, and that "suspicious" contacts should be kept under observation. One child who only had the Hofmann bacillus died in a few days. Moreover, other cases in which this bacillus only was found had mild cardiac symptoms when under observation, just as if they had suffered from a slight attack of true diphtheria.

CAUSES OF DEATH.

Barbier (*Annales de Médecine et Chirurgie Infantiles*, September 1902) studies some of the "Causes of Death in Diphtheria." The two causes especially considered are cardiac thrombosis and tuberculosis. He is inclined to believe that the former is not sufficiently acknowledged as a most frequent cause of death. In his opinion toxic myocarditis and paralytic conditions are frequently blamed for disasters which would, if a necropsy was made, be found due in reality to a thrombosis. He has looked for clots in all his autopsies, and during the year 1901 found that out of 45 examinations of fatal cases of diphtheria ante-mortem thrombi were present in no less than 23. In the following year thrombosis was noted in 14 out of 26 necropsies. This gives a total of no less than 52 per cent. The clots were most frequently found in the right cavities, either in the auricle, in the ventricle, or in both. In 7 cases only were they noted on the left side. The right auricle appeared to be the most usual site. The clots in all cases were fibrinous, of a yellowish-white colour, and more or less stratified. They were hard, resistant, and elastic, and firmly adherent to the walls of the heart. They were manifestly formed, then, during life; they were in no sense of the word the result of the death-agony. The patients, as a rule, died suddenly after presenting certain symptoms which had no connection with the process of death.

In 19 cases a bacteriological examination was made of the clots. In one case the result was completely negative: in the other 18 the Bacillus diphtheriæ was present alone in 3, and combined with the streptococcus in 3 more. In the remaining 12 cases, streptococci, staphylococci, or both combined, were found to be present. As to the cause of the condition, the writer does not commit himself. Whether it is to be regarded as the result of a septicæmia or of a pure diphtherial intoxication must remain doubtful. But he reminds us that on the tonsils and on mucous surfaces the fundamental action of the diphtherial ferment seems to be to produce fibrin, and this fact may offer an explanation of the condition.

Cardiac thrombosis is an accident of convalescence, that is to say, of the period after the disappearance of the membrane. It is surprising to find that the fifteenth day is spoken of as being the average limit of hospital observation, though we are not so astonished to learn that cases terminate fatally afterwards of this complication in their own homes. In British hospitals even the mildest case of diphtheria is seldom discharged before four weeks have elapsed. The symptoms presented by the patient most frequently were found to be a pallor of the face and chilling of the extremities. The child remained motionless on the back, and in some cases cyanosis of the lips was noted. The pulse became thready, small, quick, and irregular. There was nothing characteristic on auscultation. After the above symptoms came the dreadful distress, often accompanied by restlessness, which frequently preceded death. Consciousness was present to the end. The prognosis of such cases was, of course, most serious, and the effects of treatment *nil.*

It is certainly not a little surprising that so many of these dreadful cases of heart-failure should be found to be in connection with ante-mortem thrombosis. Most of us will agree with Barbier that many such cases are attributed to myocarditis or bulbar paralysis. The paper would have been more valuable if the condition of the heart-muscle had been noted in each case, and, further, if the presence or absence of paralysis in any situation during life had been remarked. Are we to regard this thrombosis as a primary condition ? Or could not the changes in the muscle and innervation of a failing heart predispose to such clots, even if they depend on bacilli and their products ? The question is, of course, academic to a large extent, for the patient dies in any case in the vast majority of instances.

TUBERCULOSIS IN DIPHTHERIA.

On the question of tuberculosis in diphtheria Barbier gives some interesting facts. In forty-five necropsies tubercular lesions, either of old standing or of recent date, were found, in eighteen cases, no less than 40 per cent. In twelve of the cases recent lesions were found, although some of these presented also old deposits. This suggests two questions :—firstly, what is the action of diphtheria on the evolution of tuberculosis? The fact that 25 per cent. of fatal diphtheria-cases have presented recent lesions which must have developed concomitantly with the diphtheria, and that no less than 53 per cent. of the cases in which tuberculosis was latent on admission showed fresh lesions on the post-mortem table, would go to prove that diphtheria lights up tuberculosis. The second question is, what influence has tuberculosis on the course of diphtheria? Barbier has a strong impression that the diphtheria is aggravated, and that cardiac symptoms, apart from those of thrombosis, are more frequent and severe.

UNILATERAL PARALYSIS OF THE PALATE.

Aubertin and Babonneix (*Gazette des Hôpitaux,* November 20, 1902) report three cases of unilateral paralysis of the palate consecutive to involvement of one side of the throat only. In two of the cases the false membrane was limited, or nearly so, to the right side of the fauces and in both extended over the right side of the palate. The paralysis came on early —in the second case before the throat was clean. The palate was immobile, or nearly so, on the right side, and the uvula was drawn to the left. In pronouncing the vowels A, E, I, and in making a movement of deglutition these symptoms were accentuated. In the second case it was noticed that, on swallowing, fluid regurgitated through the right nostril only. The vocal cords were unaffected. Later on, when the unilateral paralysis was less marked, though still limited strictly to the one side, paralysis of accommodation affected both eyes equally. The third case is not so clear, as the membrane, involving the whole left side of the fauces, was also present on the right tonsil. A paralysis appeared a month later, most marked on the left side, though the right side was also slightly affected. The uvula, on attempted movement, deviated strongly to the right.

Additional interest is given to this paper by three cases extracted from those observed by Colin and Acker, which are added in full to those seen by the writers. The conclusion is that paralysis is found most frequently on that side of the throat which has been most affected by the membrane. Cases, however, have been reported where a patient, whose throat was affected equally on both sides, suffered from a unilateral paralysis only. If all cases of palatal paralysis were examined carefully, it would probably be noted that many of them are merely unilateral. It is only exceptionally that, as in the second case reported, attention is drawn to a particular side by fluid returning exclusively through one nostril. Otherwise a unilateral paralysis gives precisely the same external signs as a double one. When a unilateral paralysis exists, it very seldom spreads to the other side of the palate. A generalised paralysis, affecting other parts of the body, may follow it without the healthy side of the palate becoming paralysed. Sometimes when a generalised paralysis exists, it may predominate on the side on which the palate is affected, as in a case of Acker's quoted by the writers. All this goes to prove that there is a very intimate connection between the site of the original inoculation and the resulting paralysis.

INTRAVENOUS INJECTION OF ANTITOXINE.

Perhaps the most original and useful contribution to the year's literature on diphtheria is the article by Dr. Louis Cairns (*Lancet*, Dec. 20th, 1902) on Intravenous Injection of Antitoxine. He had previously suggested this method of treatment in plague with Yersin's serum, and it struck him that the good results so obtained would also ensue in diphtheria, when the analogy of the pathology of the two diseases is considered. He therefore tried the treatment of diphtheria by this method at the Belvidere Hospital in Glasgow, and had every reason to be satisfied with the results, which anyone who reads his cases impartially will admit are absolutely brilliant. In the first place he argues in favour of very large doses of antitoxine, such doses never doing any harm to the patient; while it is only reasonable to suppose that the more serum injected the more toxine is neutralised. But even with these large doses, for instance, 20,000 or 30,000 units, a certain number of severe cases show no improvement at all, if the antitoxine is only given subcutaneously. This he attributes either to a selective influence on the part of the glands in filtering out the active constituents of the antitoxine, or to the fact that there are a definite chemical action and reaction between toxine and antitoxine, neutralisation going on more quickly in concentrated solutions than in diluted ones. We may add that it would also seem probable that the antitoxine, when obliged to pass through the glands, must depend very largely on the strength of the circulation which is notoriously enfeebled in severe cases of diphtheria, and that it probably comes through so slowly as to be in a very high dilution in the blood and tissues. Cairns points out that the tendency of the bacilli is to spread all over the system. It is now recognised that most cases of bronchopneumonia, complicating laryngeal diphtheria, depend primarily on the Klebs-Löffler bacillus. Moreover, everything in the anatomical construction of the lung seems to favour the rapid absorption of toxine. The amount of toxine absorbed in such cases must be very great, and it needs correspondingly large doses of antitoxine to counteract it. Bronchopneumonia is admitted to be the most frequent cause of death after a tracheotomy.

It was in such a case that an intravenous injection was first tried, with most encouraging results. The patient, a little girl of two years of age, was admitted pulseless, with cyanosed lips, livid, and cold at the extremities. On auscultation the heart was found to be beating 185 times per minute. The respiration was very difficult, shallow, and no less than 60, a rate certainly suggesting a pneumonic lesion. The laryngeal obstruction was almost complete. Tracheotomy was at once performed, but, though the distress in breathing became less, the lividity of the patient was unaffected. Bronchopneumonia having been diagnosed, 26,000 units of antitoxine were injected into the right median basilic vein. For two days the patient continued to be desperately ill, though the pulse was readily counted. The urine, on boiling, was solid with albumen. On the third day after injection, a marvellous and steady improvement was first noted. The lividity decreased concurrently with a free expectoration of mucus through the tube. The temperature fell steadily, and the pulse and respiration with it. The tube was removed on the eighth day. Convalescence was uninterrupted. Details of three other equally remarkable laryngeal cases, complicated by bronchopneumonia and tracheotomised, are given. In another case, in spite of an injection subcutaneously of 18,000 units on admission, bronchopneumonia developed three days later. The breathing became worse, tracheotomy was performed, and 22,000 units injected intravenously. There was immediate improvement, and the case recovered.

In malignant cases, with collars of glands round the neck and with especially filthy throats, the new treatment was found to be equally successful. Taking the results altogether, Cairns claims, firstly, that the signs of marked toxæmia rapidly disappear; secondly, that the glands subside very quickly; and thirdly, that the restlessness, so marked in broncho-pneumonic cases, ceases almost immediately after injection. In all, fifty consecutive cases are dealt with in the figures given. Of these, twenty were considered severe enough to make it worth while trying an intravenous injection. The cases were at least of average severity. The death-rate was 6 per cent. only, an astonishingly good figure. Thirty-one of the fifty cases were laryngeal and seven nasal. Fifteen were complicated with bronchopneumonia, and in seventeen it was found necessary to perform tracheotomy. The death-rate of this operation was only 5·8 per cent.

The indications given by Cairns for this method of injection are malignancy of the disease, involvement of the lungs, a moribund condition on admission, and lastly, marked toxic symptoms. The technique of the operation is not given. The initial doses vary from 20,000 to 30,000 units. Every one interested in diphtheria will admit that there are many apparently hopeless cases in which such a treatment might be of value. We all know a class of case which our experience leads us to regard as hopeless from the start. In these cases the intravenous injection should, I think, always be attempted. I have, at the Edinburgh City Hospital, latterly made a practice of using it in desperate cases. In some improvement has been very marked. It is unfortunate that Dr. Cairns did not have a real hæmorrhagic case with purpuric spots on the skin in his series. These are the most invariably fatal of all diphtherias. I may say that one of this type to whom I gave an intravenous injection lived longer than any case of the kind I have yet seen, and there is absolutely no doubt it benefited greatly by the treatment. Had it come under treatment a day earlier I believe it would have been saved. My experience, then, of intravenous injection leads me to recommend it cordially, and I look forward with interest to Dr. Cairns' next series of cases, which it is to be hoped he will also publish.

LAVAGE OF THE BLOOD IN DIPHTHERITIC PARALYSIS.

Schoull (*La Presse Médicale*, Juin 13, 1903) has a communication on Lavage of the Blood in Diphtheritic Paralysis. The patient was a man of 31 years, who on the fifth day of an attack of diphtheria had paralysis of the palate. A month later, when admitted to hospital, there was also paralysis of the œsophagus, complete paraplegia, paræsis of the upper extremities, ptosis, and cardiac pain. A few days later the condition was even more aggravated, and the bladder and rectum became paralysed. The respiration also became difficult. Injections of strychnine, caffeine, and ether were tried without result. Inhalations of oxygen also failed to relieve the patient, who appeared hopeless. Schoull then applied a large number of leeches in the neighbourhood of the anus, and injected a litre of Hayem's serum to "wash the blood" as it were. This gave a marvellous result. The patient, in a dying condition in the morning, appeared out of danger in the evening. Next day the improvement was still marked. A month later the patient left hospital completely cured. Schoull thinks this treatment might be tried with advantage in cases of much less severity.

INTUBATION.

McCollum (*Archives of Pediatrics*, October 1902) has much to say in favour of intubation of the larynx. It is curious that, although, as we shall point out shortly, this method is becoming more frequent in Great Britain, as a nation we should remain practically alone in preferring tracheotomy. McCollum prefers the O'Dwyer tube to the Bayeux pattern, thinking that the only advantage of the latter is its ready enucleation without an extractor. On the other hand, his experience is that it is more readily coughed up, which I may here note is not the case in my experience; and I am accustomed to use both types of tube indifferently. Indeed, if anything, I had an idea that the O'Dwyer pattern was coughed up more frequently. M'Collum found that with the nasal method of feeding intubated patients he had too many cases of resulting middle-ear disease. He has, therefore, discontinued it in favour of a soft rubber œsophageal tube. It may here be noted that, in the discussion which followed Dr. McCollum's paper, Dr. Jennings thought that artificial feeding need seldom be resorted to, and that a child will suck very well with its head over a table, or with the foot of the bed raised (Casselberry's position). Fairbank (*Lancet*, June 20, 1903), in a paper to be alluded to later, prefers feeding by the mouth slowly and carefully with a spoon, and thickening the milk with arrowroot if there is too much coughing. He thinks that Casselberry's position is unnecessary, and seldom uses nasal feeding except occasionally after the tube is removed, when there is not infrequently a temporary difficulty in swallowing. I myself am accustomed to feed such cases partly by the rectum and partly by the mouth, according to their ability to swallow. Semi-solids are often taken best.

McCollum believes, contrary to most writers on the subject, that adults can be intubated with advantage. He has had six or eight adult patients so treated, and he is convinced that stenosis can be more easily relieved by intubation than by tracheotomy. One of his patients was a man of sixty in whom tracheotomy would probably have been difficult. His experience of intubation goes up to the remarkable number of 1,200 cases, with a death-rate, including all causes of death, of 40 per cent. If, however, the cases which, locally relieved, died subsequently of paralysis, and the cases dying within twelve hours of admission are deducted, his percentage death-rate is only twenty-five, a very satisfactory figure. He compares it favourably with the tracheotomy death-rate of the London and Glasgow hospitals, which is about 35 per cent. This is hardly fair, as it is, I think, not quite the case, as he asserts, that cases admitted apparently moribund are not operated on in these hospitals. On the contrary, the tendency is always to operate, and no doubt if the death-rate of these hospitals were corrected by deducting the moribund and paralytic cases, the British statistics would be practically the same as his own. Personally I have always believed that it is far safer to judge a hospital by its laryngeal death-rate rather than by its operative death-rate. Some operate on a class of cases which others leave alone with equal success. One of the points in favour of intubation, to my mind, is that one can postpone it longer than one can a tracheotomy, and, after the use of antitoxine, it is always worth while giving it a chance before interfering. McCollum, while admitting that tracheotomy is obviously to be preferred in country districts, where, if a tube is clogged or coughed up, a doctor is not readily found, believes that in town practice, as well as in hospitals, intubation is much superior to tracheotomy. Its advantages are that there is very little shock, that parents consent much more readily to the operation, and that the chances of a subsequent bronchopneumonia are much less. Retained tubes are not frequently noted; in fact, he has only seen one case, and in that the tracheotomy-tube, which was subsequently found necessary, is also still retained. I may say that, in my own experience, very much less extensive than that of McCollum, I have only met with one such case, and in that also the tracheotomy-tube cannot be dispensed with. McCollum usually leaves a tube in three or four days. Occasionally only has it to be replaced.

Josias and Tollemer, in the paper (*Archives de Médecine et Chirurgie Infantiles*, November 1, 1902) already quoted, prefer the Froin pattern of tube which, so far as I am aware, has not found its way to this country. They give the useful warning that these tubes are somewhat large for the ages marked on them, and that it is safer to use a smaller size. These tubes appear to approximate more to the O'Dwyer than to the Bayeux type. They have a special arrangement for extraction with the finger, but most people who use them seem to prefer to use an ordinary extractor. It is interesting to find that, in cases where it is difficult to get the patient to dispense with the tube, Josias and Tollemer have found it most advantageous to alternate the Froin tube with that of Bayeux. I am accustomed to do the same thing in such cases, using an O'Dwyer's and Bayeux tube alternately. The points of pressure are probably different enough in the two patterns to give rest, as it were, to any part of the mucous membrane which may be threatening to ulcerate, and it is usually ulceration which prevents us from finally getting the tube out.

It is satisfactory to find that the Great Ormond Street Children's Hospital has taken to intubation. Fairbank (*loc. cit.*) reports a series of 33 cases. His percentage mortality was 27, a figure which includes cases admitted practically moribund. He emphasises the danger of opening the mouth too widely with the gag, and he prefers the recumbent position, surely the right one in a disease like diphtheria, in which it is so necessary to keep the patient flat. Unlike most writers he leaves the string in position. In this I also cordially support his views, as I have never found the retention of the string any inconvenience, if the child's arms are in pasteboard splints, a method which I notice he also favours; and on the other hand the string enables a nurse to pull out a blocked tube at once. The string is fastened to the cheek by strapping. In one of Fairbank's cases the string was bitten through. This may be, in my experience, avoided by the use of banjo-string, which is guarded with fine wire and is much more difficult to bite through. We have already noticed Fairbank's views on the method of feeding.

Although, I think, there is some doubt whether tracheotomy is not more suitable in private practice, there is every reason to believe intubation is the best operation in hospital. In America, even in private practice, the cutting operation has been practically displaced. As McCollum says in his paper, it is remarkable that in the French hospital named after Trousseau, the greatest opponent of intubation, all laryngeal cases are now treated by this method. In fact the operation seems to be in France, as in America, the operation of election. Much the same is the case in Germany. The time has come when a much more extended trial should be given it in this country.

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A REVIEW OF RECENT WORK IN THE FIELD OF VENEREAL DISEASES.

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TREATMENT OF SYPHILIS BY MERCURIAL INJECTIONS.

PROFESSOR JULLIEN, who has for the past quarter of a century been an advocate of subcutaneous injections of mercury in syphilis, still considers that this method is preferable to any other form of treatment, and that it ought to be the rule and not the exception. (*Journal des Malad. cutan. et syph.*, August, 1902.)

The action of the drug can best be exemplified by studying the process of calomel-injections. On examining the encysted mass of this substance it is seen to diminish from the periphery to the centre until its complete disappearance, this phenomenon being due to the conversion of the calomel into corrosive sublimate.

From the theoretical point of view, injections of soluble salts constitute an ideal form of treatment, since the quantity of the drug considered necessary can be introduced daily according to the urgency of the case, and no excessive strain is put upon the powers of absorption or of elimination. But many patients cannot afford either the time or the expense necessitated by such frequent visits, and it is only with a certain class of cases that this method is to be recommended. These daily injections are appropriate to dyspeptic subjects, and also in cases of renal disease, since their slowly-progressive action and the supervision it enables the surgeon to exercise on the functions of the kidneys are a guarantee for the safeguard of these organs.

It is contra-indicated in cases of extreme urgency, where it is necessary to strike a decisive blow, in order to save at any cost a vital organ, the integrity of which would be jeopardised by the least delay.

Which salt should be selected? The bichloride, the salt originally recommended, in aqueous solution with a small quantity of chloride of sodium, sets up some local irritation, and requires a considerable dilution to prevent the dose of one centigramme from giving rise to considerable pain. Under the form of peptonate of mercury this salt is better tolerated, but this substance is difficult to prepare and liable to decompose. The cyanide in doses of 1, 2, or even 3 centigrammes is reliable in its action, but occasions both pain and swelling. The benzoate is less active, requires a dose of at least 2 centigrammes, and necessitates the addition of cocaine in order to mitigate the pain following its introduction. Much has been said in favour of the cacodylate, but this is not a pure mercurial salt, but a cacodylate of iodic. hydrarg. The succinimide has been warmly recommended, and, without being absolutely painless, is certainly excellent from the point of view both of its local and general action. One of the latest introductions, the "hermophenyl," is remarkable for the very little irritation it produces, but requires a larger dosage; the soluble salicylate in doses of one centigramme is active and well tolerated; the biniodide in oily solution is considered by some as a sort of panacea, but it is not superior to other compounds, and is, moreover, productive of considerable pain. These watery injections have the further advantage that they can be mixed with a varying amount of artificial serum according to the requirements of the case.

As regards the insoluble salts, one of the most remarkable features of this method of introducing the drug was the rapidity with which it was absorbed into the system, since mercury was found to be present in the urine two or three hours after the injections, and in the saliva four or five hours after. Abscesses, which in former days frequently supervened, are now only very rarely encountered. Among the substances recommended might be mentioned calomel, yellow oxide of mercury, thymol-acetate, salicylate, and the "grey oil." Salicylate of mercury is remarkable for the way in which it is tolerated by the muscular and cellular tissues; there is no fear of any inflammatory reaction, and the formation of indurated nodules is exceptional; no stomatitis follows, and its action is very certain, though somewhat slow; two injections of 6 centigrammes may be made weekly, and it can be employed for a long time without giving rise to any intolerance.

The "grey oil" is more active, and, although well tolerated, is less so than the salicylate; often the patients complain of a feeling of bruising, sometimes amounting to actual pain; its action takes longer to show itself, and is not manifest till after the second injection. One course of grey oil, consisting of six injections at the most, four at the least, at eight days' interval is sufficient to cause all the ordinary symptoms of syphilis to disappear; and this course renewed two, three, or even four times a year, according to the powers of resistance of the patient and the intensity of the symptoms, will suffice to control the disease during the first years of its existence, whilst later one injection monthly will ensure a favourable termination of the case.

F. Trémolières (*La Presse Médicale*, March 25, 1903) also advocates intra-muscular injections in syphilis.

This treatment is gradually coming into vogue, and deservedly so from the many advantages it possesses; it renders the absorption of mercury more certain, more rapid, and more marked than in other methods of treatment; it does not disturb the digestive tract as when the drug is administered by the mouth, and is not, like inunctions, uncertain and disagreeable. True, it entails certain inconveniences which are inseparable from mercurial treatment, such as diarrhœa, stomatitis, and albuminuria, whilst in addition it may give rise to pain and localised swellings, but any further inconveniences may be avoided by the skill and care of the operator.

The hypodermic treatment resolves itself into two different methods; frequent injection in minute doses of soluble preparations, or the occasional injection of insoluble salts in larger quantities. For the injection of soluble salts a small quantity is introduced daily, and is at once absorbed and eliminated, the treatment being in proportion to the intensity of the disease. But the daily use of the injections is irksome to the surgeon and costly to the patient, and the pain, though inconsiderable in itself, may by its frequent repetition become almost unbearable.

The employment of the insoluble salts is free from these drawbacks, for here a certain amount of mercury is introduced at much longer intervals, and is absorbed slowly and continuously; sometimes the effect of this treatment is intense, rarely the material injected becomes encysted and remains inert; in case of mercurialisation the effect of the drug cannot be controlled or modified, and this is the principal objection to this plan. The pain and the localised induration consequent upon the injection are reduced to a minimum by the employment of a good preparation and by the careful carrying-out of the technique. The mercurial salt should be of a definite chemical composition, and should be easily assimilated by the fluids of the organism by which it should not be precipitated. It should be dissolved or emulsified in a carefully-sterilised vessel of neutral or alkaline reaction; the less the quantity of the substance injected, the less pain is experienced. A syringe easily taken to pieces, and capable of thorough sterilisation, should be employed with platino-iridium needles from 4 to 6 centimetres in length. The needle should be thrust into the midst of the muscle in the upper third of the buttock, or into the retro-trochanteric region. In every case the surgeon ought to examine for albuminuria and glycosuria; he should inspect the condition of the mouth, and should commence with small doses so as to test the susceptibility of the individual.

Under the influence of mercury the general health improves, the excretion of urea increases, the nitrogenised products are elaborated in greater quantities, and the patient gains weight and strength. These results are obtained sooner by the hypodermic method than by internal ingestion, seeing that the absorption of mercury is far more rapid from the former procedure; in fact, mercury appears in the urine two or three hours after injections, whilst its appearance is delayed for twenty-four hours after inunctions, and for three to five days after the administration of pills.

The percentage of mercury in the different salts regulates their therapeutic effects, but the preparation the most rich in mercury may give rise to great irritation, while one of less strength may be better tolerated and so larger doses may be permissible.

Of the insoluble preparations the best appear to be calomel, proto-iodide, salicylate, and grey oil. Calomel, it is true, presents serious inconveniences; it is very painful, liable to be followed by intense inflammatory reaction, by swelling, by induration, and sometimes, in spite of all precautions, by abscess. But it is a heroic remedy, with a remarkably rapid and powerful action; it may not be indicated in the prolonged and methodical treatment of syphilis, but it is appropriate in all cases where it is necessary to strike forcibly and quickly, such as phagedænic chancres, malignant precocious syphilis, dry skin-lesions, cutaneous or mucous ulcerations, visceral and nervous syphilis.

Of the numerous formulæ that of Balzer may be recommended:—Calomel, 1 gramme; oil of vaseline, 10 cubic centimetres.

It is the one best tolerated by the tissues, and gives rise to the least local reaction. Five or six centigrammes injected every eighth day for five or six weeks produce the most satisfactory results; it is most important that the preparation should be fresh, and mixed, if possible, on the day it is to be utilised.

Without possessing the same powerful influence as calomel, the proto-iodide of mercury appears to have an energetic action on syphilitic symptoms, whether secondary or tertiary. It is, moreover, less painful, provokes less inflammatory reaction, and is in fact better tolerated.

The basic salicylate is well borne, is painless, and is not followed by toxic symptoms, but its action is slow and feeble; it is not sufficient to cope with any severe manifestations, but it may with advantage be employed for continuous treatment.

The grey oil, with 40 per cent. of mercury, seems to be best of the insoluble preparations; its curative action is remarkable, it is well tolerated, is usually painless, is not followed by any inflammatory reaction, and does not give rise to any induration. It does not cause diarrhœa, and in patients with good teeth and in general good health does not set up any stomatitis. Its efficiency and its harmlessness render it appropriate in the chronic intermittent treatment of syphilis by injections. Certain precautions, however, should be observed in its employment; the emulsion should be of equal consistency throughout, and perfectly sterile, as, without these conditions, disappointments may be met with, for which the treatment is undeservedly blamed. As a general rule, injections of 6 or 12 centigrammes of mercury should be given weekly in a series of 6 or 8 injections, separated by a two-months' interval.

The soluble preparations are more numerous than the insoluble, those most in favour being the biniodide, the lactate, the neutral salicylate, and the benzoate.

The biniodide is employed either in oily or in watery solution. The former has been used in doses of 30 milligrammes to 2 cubic centimetres of oil, and has produced excellent therapeutical results without causing diarrhœa, stomatitis, or other symptoms of intolerance, though producing considerable pain and often induration. The aqueous solution of the biniodide is rendered soluble by the addition of an equal quantity of iodide of sodium; its introduction is usually painless, and produces no inflammatory induration; it is, nevertheless, remarkably effective, and in a dose of from 4 to 6 centigrammes it rapidly disperses grave symptoms, without giving rise to any toxic phenomena. Diarrhœa may appear, but this will yield to treatment. The lactate of mercury in a daily dose of 3 centigrammes produces good results, but even if it does not set up symptoms of intolerance, it is painful and often occasions inflammatory indurations; to remedy these inconveniences a double lactate of mercury with cocaine has been devised.

The neutral salicylate of mercury is equally efficacious; a series of 20 to 25 injections of 2 centigrammes causes the disappearance of all the specific symptoms.

Benzoate of mercury gives rise to very little pain, no swellings, no diarrhœa or stomatitis; it should be pure and freshly prepared, and dissolved in artificial serum; the solution injected in a dose of 2 or 3 centigrammes is well tolerated.

Leredde (*The Medical Press*, Oct. 29, 1902) emphasises the necessity of treating the graver complications of syphilis by means of mercurial injections, and also of considerably increasing the doses of mercury at present employed. Injections are the only means we possess of administering a definite quantity of mercury. Experience has proved that the therapeutical effect of proto-iodide pills is inferior to that of a weekly injection of 10 centigrammes of calomel, and that the therapeutical effect of inunction in a daily dose of 6 grammes is almost invariably inferior to that of the calomel-injection. It is not to be supposed that a patient who takes daily 10 centigrammes of proto-iodide absorbs the whole of the salt, or that half the amount rubbed in by the inunction-method is taken up by the system; and it may be taken for granted that it is only by means of injections that a known quantity of mercury may be introduced into the organism, and no other procedure is open when we wish to obtain the maximum curative effect of mercury. Since the therapeutical efficacy of a mercurial compound depends solely on the quantity of mercury introduced into the organism in a given time, it may be well to enumerate the various mercurial compounds in general use, giving the quantity of mercury contained in each:—

Mercurous chloride (calomel) -	-	84·92 per cent.
Cyanide of mercury - -	-	79·32 „
Mercuric chloride (sublimate) -	-	73·80 „
Mercurous iodide - -	-	61·16 „
Benzoate of mercury - -	-	45·25 „
Mercuric iodide (biniodide) -	-	44·05 „

This shows that of the soluble salts, the cyanide and sublimate, contain nearly twice as much mercury as the benzoate and biniodides. Of all the salts in general use calomel is the richest in mercury and the most effective from the therapeutic point of view. Each injection is followed by a considerable reaction, accompanied by rise of temperature and lumbar pain lasting for at least three days, during which time the greater proportion of the calomel introduced into the muscle is absorbed. Injections of grey oil provoke very little reaction, and are slowly absorbed as is proved by the late supervention of stomatitis. The question of dosage should occupy a prominent place in the consideration of mercurial treatment; all cases of syphilis are not cured by the same doses, but if curative effects do not follow a known dose of mercury, it is only necessary to increase the dose in order to effect a cure. We often meet with syphilitic lesions of the viscera and nervous system, which are believed to be refractory to anti-syphilitic treatment, but which might be cured by the employment of larger doses than those usually resorted to. Injections of the insoluble are believed to give better results than those of the soluble salts, though probably this is a fallacy, for at present we employ injections of soluble salts in only small doses, whereas with the insoluble salts a much larger quantity of mercury is introduced, and so their activity is more pronounced. The dangers of mercurial poisoning are not really great, for the toxic dose is not approximate to the therapeutic. Mercurial stomatitis depends essentially on the state of the buccal mucous membrane, and those whose gums and teeth are in good condition at the commencement of treatment are able to tolerate large doses of mercury without stomatitis. The treatment of grave syphilitic troubles may then be modified by increasing the quantities of soluble salts or of calomel introduced into the organism; we may employ benzoate of mercury in daily doses of from 5 to 8 centigrammes, or the biniodide in doses of 6 centigrammes. If the sublimate or cyanide are employed, it must be borne in mind that these salts contain more mercury, and should be administered in doses less by one half than those of the biniodide and benzoate. Generally, when it is desired to give mercury in large doses, it will be preferable to inject the soluble salts, because it will be easier to stop the absorption of the mercury should stomatitis or general phenomena of a toxic origin supervene. Large doses of mercury are indicated when that drug is administered as a means of diagnosis; when experience shows that the ordinary doses are not curative; in cases where a normal dose might be insufficient to prevent irremediable mischief. In lesions of the nervous system the maximum doses should always be employed, as in tabes, general paralysis, and other affections of the nervous system described by Fournier as parasyphilitic. The failure of treatment in such cases may be due to the fact that general paralysis and tabes are not invariably of syphilitic nature and origin, that the treatment is usually commenced too late, and that the doses employed are inadequate to the gravity of the disease. In conclusion, mercury may be employed more energetically than has usually been done for the cure of grave syphilitic troubles, without any actual danger to the patient, and in this manner results may be obtained which were formerly impossible.

A MODIFIED FORM OF "GREY OIL."

Heidingsfeld (*Indian Medical Record*, Oct. 29, 1902), recommends, for intra-muscular injections in syphilis, the following formula as an improvement on the grey oil introduced by Lang :—

.—Lanolini, 2 parts.
Hydrargyri bidistil, 6 parts.

Mix till the complete disappearance of the mercury, and add—

Liq. albolene, 2 parts.
Sig.—Inject 1 grain every three or four days.

This preparation can readily be drawn into a hypodermic syringe, the needle applied, and successive doses measured off by means of the retaining-screw ; one injection, containing 1 grain of mercury, should be administered every four days for two months. A ⅞-inch 22-gauge steel needle is employed, which ensures that the injection is deposited deeply into the subcutaneous fat ; injections into the muscles were painful, and not well tolerated. Pain and induration seldom followed the injections, and in only one instance out of 2,000 did abscess follow. Marked improvement was noted in all the cases ; material gain in weight, rapid amelioration of symptoms, improvement in appetite. Cases that had shown intolerance to internal medication and inunction promptly responded to this form of treatment. In the vast majority of cases the treatment is well borne, but in a few it gives rise to such pain as to render other methods desirable. It is best to begin the treatment with a minimum dose, gr. ¼ to ½, and gradually to increase to gr. 1 or more, in accordance with the degree of tolerance manifested by the patient.

INJECTIONS OF AQUEOUS SOLUTION OF BINIODIDE OF MERCURY.

Emery and Druelle (*La Presse Médicale*, February 11, 1903) give the result of their experience in this plan of treatment for syphilis at the Saint Louis Hospital in Paris. To obtain a suitable solution of the biniodide, they recommend the addition of an equal weight of dried and purified iodide of sodium, and also a small quantity of chloride of sodium. The strength of the solution used was 2 centigrammes to 1 cubic centimetre. The cases selected were those in which the results of the treatment could not be contested, large tertiary syphilitic gummata, lichenoid and acneïform papular eruptions which had resisted treatment, and chancres of long duration. The dose usually given was 2 or 3 centigrammes daily, so that the action of the drug should be as effective as possible, and that the limit of tolerance should be reached; the daily dose of 2 centigrammes was well borne, and produced good results, but required a rigorous supervision of the patient. Amongst some of the drawbacks of the treatment must be noted the diarrhœa, which not infrequently supervened, and which occasionally necessitated an interruption of the treatment for a few days. Ptyalism also might be set up, but in no instance was it serious, and it invariably disappeared on stopping the treatment for a few days. The pain following the injection is as a rule trifling, and was less after deep than after superficial injections; often one injection may give rise to pain, while the 10 or 12 consecutive doses are quite free from this symptom. It should be noted that the patients treated by this method were the subjects of serious and intractable symptoms, lesions ordinarily treated by iodide of potassium and mercury combined.

A CASE WITH COMMENTS.

A CASE OF SPLENIC LEUCHÆMIA IN A YOUNG CHILD.[1]

By JOHN McCAW, M.D., R.U.I., &c.,
Senior Physician to the Belfast Hospital for Children.

ISABELLA H., age 1 year and 8 months, was admitted into the Belfast Hospital for Sick Children under my care on February 20, 1903.

Family History.—The father and mother are living; both are strong and enjoy average good health. No history of syphilis in either parent could be obtained.

The family consists of four children as follows:—The first child was born at full term; it was apparently healthy and well-developed, but died when three days old; no cause could be given for its early demise. The second child was born at full term and was well-developed. She is now a strong healthy girl of five and a half years. The third child was born at full term and was strong and well-developed. At the age of 10 months he contracted whooping-cough, and this, becoming complicated with broncho-pneumonia, ended fatally.

The fourth child is the subject of this paper. She was born at full term and was healthy in every respect. She was breast-fed for one year, and developed into a large and strong child. The present illness is the first from which she has suffered. The mother states that the child was able to toddle about until three weeks prior to the time when she was brought to hospital, about which period she was observed to become weak on her legs and to stagger much when walking. About the same time she became drowsy and apathetic. A week later her abdomen was seen to be enlarged and the glands of the neck to be swollen, but she did not complain of any pain. Dr. Hogg, the family physician, was consulted; and, acting on his advice, the child was brought to the hospital.

State on Admission.—The patient is a well-developed and bulky child for her age, her weight being 1 st. 13½ lbs. No sign of either rickets or scurvy is present, and her appearance is that of a full-blooded rather than an anæmic child. On examination the chest organs are healthy.

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[1] Read before the Society for the Study of Disease in Children on May 15, 1903.

The Glands in the neck, in the axillæ, and in the groins are considerably enlarged; they feel hard, are freely moveable one upon the other, and show no sign of softening anywhere; they are not tender on free manipulation.

The Abdomen is enlarged generally, and this enlargement is found to be due to the enormous size of the spleen, and also, but to a less extent, to the large size of the liver.

The Spleen is easily visible as a large mass which moves up and down with each respiration. On palpation it is found to extend forwards to the umbilicus, while its free margin can be readily traced downwards as far as the pelvic brim. It is very hard and dense to the feel, and is evidently tender, as any manipulation of it is resented. The surface is uniformly smooth, and the notch is very distinct.

The Liver extends downwards for 1 inch below the level of the umbilicus and almost meets the spleen in the middle line. It is not so hard, nor is it so tender on palpation, as is the spleen.

The Kidneys are apparently normal in size. The urine presents a very peculiar appearance. It is cloudy when passed, but when allowed to stand until it becomes cold it becomes quite thick and looks exactly like butter-milk. The first specimen which was shown to me struck me at once as being chylous urine. On examination the condition of the urine was found to be due to an enormous deposit of salts, among which were found numerous crystals of oxalic acid; there was a distinct trace of albumen. The reaction was intensely acid; the specific gravity was 1,023, and the amount passed in 24 hours was 12 oz. The temperature on admission was 98° F., and during her stay of one week in hospital no febrile movement was recorded, except on February 22, when the thermometer registered 100·8° F. The blood was examined by Dr. Tom Houston, and the following is his report:—

Red cells - - - -	2,200,000 per c. mm.
White cells - - - -	810,000 „ „
Hæmoglobin - - -	38·8 per cent.

Differential Count.

Polymorpho-nuclear cells - - ·8 per cent.
Lymphocytes - - - - 99·2 „ „
Nucleated red cells (Normoblasts) - A few.

The lymphocytes contained a nucleus which stained deeply with basiphile stains, and the surrounding rim of protoplasm was more deeply stained than the nucleus. Many of these cells showed signs of degeneration. The low percentage of hæmoglobin is remarkable, as the clinical appearance of the little patient was not at all one of anæmia.

Two days after admission a few purpuric spots were observed on the legs and arms, and a day later a few bruise-like marks made their appearance, one over the left eye, one on the upper left arm, and one on the right thigh. These marks did not increase in size, nor did any fresh ones appear. No bleeding from either serous or mucous surfaces took place during her stay in hospital.

The child became very fretful and cried almost incessantly, for which no cause was apparent. She was taken home in a week. I was asked to see her with the family physician on the 5th of March, when the following note was made: She is now markedly anæmic and has lost flesh considerably; her tissues are soft and flabby. The temperature has remained about normal, and was 99° F. at the time of my visit. She is less fretful, but is tender over the body generally, and cries when lifted or handled. No hæmorrhages have occurred, with the exception that the motion passed on the previous day was streaked with blood. Several fresh bruise-like marks, about the size of a bean, are visible over the body, and on the right side of the chest and abdomen are numerous purpuric spots; a few also are to be seen on the legs. The spleen is, if possible, larger, and now fills the entire left side of the abdomen, extending well beyond the umbilicus and reaching down into the pelvis. It feels very hard and resistant, and is evidently very tender, as any pressure upon it causes the child to cry out. The liver remains about the same.

The child died seven days later. From the time of my visit till her death she became rapidly weaker and sank into a comatose state. Dr. Hogg informs me that her breathing became very rapid though no lung-trouble was present to account for this; and also that a considerable amount of cyanosis was developed. No hæmorrhages took place, nor was there any increase in the purpuric spots and bruise-like marks. A very noticeable feature was the considerable diminution in the size and hardness of the spleen before death. The lymphatic glands also became much smaller, and they too were not so hard to the feel. No post-mortem examination would be permitted—a circumstance to be regretted.

Treatment.—This consisted in giving arsenic in the form of Fowler's solution. The dose first given was four minims three times daily, which was rapidly increased, until she was taking ten minims at the time of death. It could not be said that any benefit resulted from its use.

REMARKS.

This case, which ran its course in about six weeks, is an example of a very acute type of splenic leuchæmia. The appearance of the child on admission to hospital was sturdy and well-developed, and no one would have imagined that she was suffering from a serious blood-disease. The examination of the blood, however, disclosed a condition which was a great surprise, and enabled the diagnosis to be made in favour of leuchæmia rather than of lymphadenoma. The extreme fretfulness and restlessness may, I think, be accounted for by the condition of the medullary substance of the long bones which was probably undergoing rapid degenerative changes. I am unable, however, to offer any explanation of the somewhat rapid diminution in size of the spleen and lymphatic glands before death.

Notes by the Way.

Protozoa as Causes of Disease.

THE tide seems to have turned at last, and the bacteria no longer exclusively monopolise the interest of researchers in the field of pathology. Several diseases have recently been ascribed to protozoa besides malaria, for which their culpability is well established. Yellow fever has been the subject of numerous investigations since the Spanish-American war, and its exciting cause is now believed by many to be of animal nature. It has been suggested that the mosquito is the principal host in this case, and man only a subordinate link in the chain of its development. Very interesting observations have been made by Councilman and his assistants on the pathology of small-pox, and here again appearances resembling protozoa have been described, as they were some time ago by Guarnieri and Pfeiffer. The malady known as dengue has also been asserted to be of protozoan origin. Time is needed to show what the truth may be as to the causation of the Sleeping Sickness of the Congo. Here two organisms have been described, as was pointed out by Professor Hewlett in our August number. One of these is a bacterium, which has been called the "hypnococcus," while the other is a protozoön, of the class called "Trypanosoma." Both are said to be found only in this disease, and their respective claims to be the true cause of the malady have yet to be determined.

In view of the importance which protozoa are now assuming in the production of disease, we are glad to welcome a little book on the subject of these minute animals,[1] written by Mr. Jackson Clarke, in which a good description is given of those which are known to be pathogenic. It was quite time that a text-book on the subject made its appearance, as so far no convenient work on these parasites was available. A second volume is promised, dealing with those members of this class—

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[1] *Protozoa and Disease.* By J. Jackson Clarke, M.B. (Lond.). Part I., pp. xx and 177. Illustr. 91. London: Baillière, Tindall & Cox. 1903. Price 7s. 6d. nett.

or perhaps we ought to say sub-kingdom—the pathogenic qualities of which are still doubtful. We may expect to find in this second volume an account not only of those reputed parasites to which we have just alluded, but also of the protozoa reported to be the cause of hydrophobia, while perhaps the much-sought-for parasite of cancer will by that time have been identified as a protozoön. Perhaps it will not.

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Cancer-Combatants.

We are pleased to publish this month an article on the subject of the causation of cancer by Dr. A. T. Brand, especially as it is to a large extent a reply to some comments of our own in a past *Note by the Way.* For this reason, we need not apologise for examining the arguments contained in it somewhat critically. We feel that some of the shafts which it contains are aimed at ourselves. At the outset we must confess that we are not quite sure whether we are to be classed with the contemptible invertebrate beings who have open minds on the subject of the causation of this disease, and who decline to rush headlong into the fray armed with self-confidence and a little knowledge; or whether we are rather to see ourselves depicted as opponents of the parasitic hypothesis—"fools and blind" indeed, who confine themselves to "stating objections, some of which are pure fallacies, while others are too frivolous to be seriously considered," or who fail to appreciate the facts of the case, but who yet subserve a useful function as barbarians and infidels, to be trounced and cudgelled by the true believers. We are inclined to think that Dr. Brand in his charity means to place us in this second class, but our conscience rather bids us see ourselves in the rôle of "dilettante agnostics" or "Gallios" who believe that nothing, or at least very little, is known as to the cause of cancer. However, we venture to differ from Dr. Brand in his view that this attitude is "calculated to delay indefinitely all progress" in the elucidation of the cause in question. Probably no single individual in the land who knows what cancer is, and is conscious that he may himself suffer from it, or see those nearest and dearest to him so afflicted, could be said not to care whether the cause is discovered or not. We merely doubt whether vigour of polemics will aid much in the search, and whether those who keep open minds are not as likely to have also open eyes, as are those who are red-hot partisans in matters in which proof is wanting.

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Facts, and their Interpretation.

As to the facts of the case, so far as they have been ascertained, we are glad to find ourselves in agreement with Dr. Brand. It is in the interpretation of them that we differ. Malignant growths appear to form their characteristic secondary deposits by implantation of cells which have escaped from the original tumour and settled in other parts of the body: hence the identity of structure which is observed for the most part between the original growth and the metastases. Dr. Brand seems to find in this an argument for the parasitic nature of cancer, whereas it seems to us that this migration of cells is something distinct from anything known to happen in the recognised infective processes, and that therefore it constitutes an argument against the infective hypothesis as applied to tumours. Dr. Brand says that "It would be absurd to suppose that pathogenesis can be fettered by analogy," which is no doubt true, and is about as illuminating as to protest that the movement of the earth is not fettered by the multiplication-table. Facts are not fettered by rules of logic, but it is well to observe these rules in arguing about them.

In default of the isolation of a parasite, and of resulting experimental enquiry, we are obliged to trust to some extent to arguments from analogy between cancer and infective processes. The argument from analogy as applied to the question before us would be somewhat as follows:—Cancer resembles infection in its mode of invasion of neighbouring tissues, in its production of secondary foci at a distance, in its (rare) conveyance from one individual to another, and in its endemic prevalence (?). Both these last points are doubtful, but we give the argument in its strongest form. Therefore, it may be said, cancer also resembles an infective disease in being due to an external agent. To upset this argument from analogy it seems sufficient to point out that cancer differs from known infective diseases in the mode in which it is spread in the body of the affected individual—a very material point. No argument from analogy—resemblance—can therefore be safely drawn between the two conditions. Of course this does not prove that cancer is not an infective disease, but merely that it cannot be proved to be so by this particular method.

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Innocence and Malignancy.

FOR practical purposes a malignant growth is one which insinuates itself into the neighbouring tissues, and which forms secondary deposits in distant parts. An innocent tumour merely increases in size, and what injury it does, is due to pressure on surrounding structures. Now as it is admitted that a malignant growth forms its metastases by detachment of cells from its periphery, these cells being carried by the blood or lymph to distant parts, it would seem that the essential difference between innocence and malignancy consists in the looseness of coherence between the cells of the latter variety of growth. Malignant tumours consist as a rule of soft material, without any limiting membrane, whereas benign growths are generally firmer, and often possess a well-marked capsule. It is impossible to see in this any fundamental difference of nature between the innocent and the malign. It would seem probable, therefore, that the same mode of causation is at work in both classes. It is not impossible that in each case it may be a parasite, but the upholders of this theory have never attempted to apply it to the simple tumours.

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Influence of Age.

DR. BRAND, like other defenders of the parasitic hypothesis, points to the occurrence of cancer in old age when the resistance of the tissues is presumably low, as an additional argument in favour of his theory. Unfortunately some of the most malignant growths occur in the young, even in infants, as for instance the melanotic sarcoma of the choroid, while true epithelial cancer is more malignant, as a rule, the younger the subject which it attacks. The vitality of the cells of the growth seems to be in proportion to the vigour of the patient, not contrariwise, as should occur in an infection.

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Infectivity of Cancer.

ONE more argument of Dr. Brand's must be noticed. He says:—"No one can deny that cancer is most intensely infectious to the individual, and it cannot be gainsaid that all diseases which are infectious to the individual are capable of communication to others, and are also of external origin." We venture to think that this short quotation contains at least two of the fallacies which Dr. Brand imputes to the opponents of the parasitic hypothesis. If we put the argument in other words, avoiding "question-begging epithets," the weakness of the reasoning is apparent. "No one can doubt that cancer spreads from one part of the affected individual to another; therefore it can be communicated to other persons." The "*non sequitur*" is apparent. We quite admit, however, that, as Dr. Brand has pointed out, there are observations which tend to show that cancer may be communicated from one person to another; it is the argument rather than the conclusion to which exception may be taken. But the rest of the quotation which we have given contains a distinct "*petitio principii.*" Supposing that cancer is communicable from one individual to another, it is still necessary to prove that it is due to a parasite. It is quite conceivable that if it consist in a disorderly growth of living cells due to intrinsic causes, not to an infective agent, these cells might be as capable of transference to a second individual as they are of carriage from one part of the infected body to another. The process might be analogous to grafting of tissue.

As a result of the whole consideration of the case, we venture to maintain our original position "on the fence." We are quite ready to accept as the cause of cancer any parasite which can be found to fulfil the conditions of proof, but in defect of such a discovery we think that the available evidence is rather against than in favour of such a mode of causation.

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Ultra-microscopical Causes of Disease.

IN order to vindicate our claim to an open mind, we are ready to admit that there may be several causes why the parasite of cancer, if there be one, has not been discovered in spite of the prolonged search which has been made for it. In the first place it may be a protozoön, and unsuitable for growth on artificial media. In defect of this means of demonstration, it would be very difficult to prove its existence. Secondly, it may be too small to be visible even by means of the highest powers of the microscope. That there are some contagia of this minute description has almost been proved, since in certain diseases, such as yellow fever, it has been found possible to convey infection by means of filtered extracts which contain no visible particles. A very interesting discussion of some of these conditions may be found in the recent numbers of *The Journal of the American Medical Association* (Aug. 15 and 22), in a paper by Dr. Ludwig Hektoen. The reason that the causal agents of syphilis, measles, and other common diseases have escaped identification, may be that they lie beyond the regions of even microscopical visibility; and cancer may perhaps be found to own a similar cause.

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Addison's Disease.

A VERY interesting paper by Dr. Adams will be found in our present number, dealing with the use of extracts of the suprarenal glands in the treatment of Addison's disease or tuberculosis of these structures. Much disappointment has ensued in the majority of cases in which this method of treatment has been adopted. It was at first hoped that the extract would give us as sure a means of combating this disease as we possess in thyroid extract for the relief of myxœdema. It must, however, be remembered that even in myxœdema there appears to come a time when the artificial supply of the necessary material is no longer sufficient, so that patients suffering from this condition, although they improve marvellously for a period, finally succumb to the malady in spite of the remedy. In Addison's disease a certain proportion of cases have been recorded as improving under the use of suprarenal extract, but as Dr. Adams' statistics show, too often this amelioration is only temporary, and the fatal result is only postponed. In a still larger number no definite improvement is observed; while there seems reason to think that in a few instances death was directly due to subcutaneous grafting of living gland-tissue—a method which is, therefore, to be given up.

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Use of Ductless Glands.

In spite of the advances made in our knowledge of the thyroid gland, and the function of its secretion, made at the end of the last century, we are still very much in the dark as to the physiology of similar structures, such as the suprarenals and the pituitary body. As to the former it seems to be established that they have a powerful influence on the maintenance of vascular tone, while the connection of pigmentation, as seen in Addison's disease, with these structures is to some extent borne out by the discovery of chromogenic substances in these little organs. Further than this we cannot at present go. Dr. Sajous has indeed elaborated a theory according to which the whole control of the functions of life resides in the suprarenal glands, and in the pituitary body, which is the nervous centre for their guidance. That they do subserve some important function is undoubted, and in the sense that they are necessary for life they may be said to act as controlling factors, but we are not yet prepared to put the other organs into quite a subordinate place. The soul was said at one time to reside in the pineal gland, which subsequent research in comparative anatomy showed to be in reality a rudimentary eye. We rather doubt the claims of the pituitary body to the exalted position assigned to it by Dr. Sajous. There seems some reason to associate changes in it with the curious disease known as acromegaly, but on the other hand it may be extensively involved in some morbid process without apparently giving rise to any symptoms. Its functions are in reality quite unknown.

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Matrimony and Refraction.

Not long ago Dr. G. M. Gould proved to his own satisfaction that Carlyle, along with many other great men, suffered from ocular defects which were accountable for much of his ill-health; and the same ingenious authority has more recently adduced evidence to show that Mrs. Carlyle was similarly afflicted. *Hinc illæ lacrimæ!* The moral is obvious. In all cases of matrimonial differences both parties should at once repair to an ophthalmic surgeon and be fitted with appropriate spectacles. This simple procedure may obviate much subsequent unpleasantness and bickering. It may even save a too trustful biographer from a flood of calumny, and protect self-advertising busybodies from themselves, by depriving them of an opportunity of exhibiting their lack of taste.

Novelties and Notices.

SOME NEW MANGANESE PREPARATIONS.

Messrs. Burroughs, Wellcome, and Co. send us specimens of some novel preparations of manganese which should be of interest to prescribers. The value of the metal in the treatment of anæmic and debilitated conditions has been long recognised, and recently attention has been called to the use of manganese in dysmenorrhœa and other uterine ailments. Hitherto the permanganate of potassium was almost the only soluble salt available as a remedy, the dioxide of manganese being probably inert. The preparations before us consist of the citrate of manganese, the double citrate of iron and manganese, and a double phosphate of the same metals. The salts are all of the "scale" type, and are readily soluble. These new additions to our therapeutic resources may be obtained in tabloid form, or in bottles containing one or four ounces of the salt, as desired.

SALICYLATE OF QUININE.

The value of the salicylate of quinine as a remedy which combines the properties of the alkaloid and of the salicylates is becoming generally recognised, and not long ago a paper in our own pages indicated the uses of this drug in a number of different ailments. A convenient tabloid containing one grain of the salt is now issued by Messrs. Burroughs, Wellcome, and Co., in addition to the three-grain tabloids which they have for some time supplied. The new form may be recommended for children, or for those who are sensitive to quinine in any form.

TABLOIDS OF IRON PHOSPHITE AND HYPOPHOSPHITE.

Another addition to Messrs. Burroughs, Wellcome, and Co.'s list of tabloids is a useful preparation containing two grains of the phosphate of iron and one grain of the hypophosphite of this metal. These salts have been recommended for use in anæmic conditions accompanied by nervous symptoms (depression, &c.), and form a useful preparation of iron in other conditions. The tabloids are readily soluble, and are sugar-coated. One to three are considered a suitable dose. They are put up in bottles of 25 and 100.

"SOLOID" ROMANOWSKY'S STAIN.

Romanowsky's stain (a combination of methylene blue and eosine) is one which has been much used for the preparation of blood-films for the demonstration of the malarial organism. It forms a useful addition to the list of dyes which are manufactured by Messrs. Burroughs, Wellcome, and Co. in "Soloid" form. Devotees of the microscope who live at a distance from a town in which there are facilities for obtaining staining reagents, must appreciate the advantages offered by the solid stains made up in carefully-weighed quantities which are supplied in the above shape. The soloids of Romanowsky's stain represent 0·015 gramme of the solid material, and are intended to be dissolved in 10 cc. of absolute alcohol. A few drops of the solution are run on to the blood-film, left for four to five minutes, and then washed off with distilled water. The corpuscles are stained red, and the nuclei of the leucocytes, &c. pink.

"MALTICO" INFANT FOOD.

"Maltico" Infant Food, manufactured by Taylor and Sons, Kingston Cross, Portsmouth, is a brownish powder of agreeable malted flavour, readily soluble, and apparently free from starch. In view of this last quality, it should, under careful medical direction, prove a useful addition to our resources in the way of infants' foods. It is stated to be prepared with milk and malted cereals, and is sold in bottles costing respectively 1s. 6d. and 2s. 6d. according to size.

"SWAN" PENS.

The "Swan" pens, made by Messrs. Mabie, Todd and Bard, are too well known to our readers to need recommendation. In view, perhaps, of the not-very-distant Christmas season, the makers of these pens have issued a new catalogue of their specialities which may be commended to those who are on the look-out for useful presents a few months hence, as well as to medical men in search of a convenient pocket pen for their own use.

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Reviews of Books.

Selected Papers on Operative and Clinical Surgery. By the late Sir WILLIAM STOKES, M.D., M.Ch. (Univ. Dub.), F.R.C.S.I., Ex-President of the Royal College of Surgeons, &c. Edited by WILLIAM TAYLOR, B.A., M.D. (Univ. Dub.), F.R.C.P.I., Surgeon to and Lecturer on Clinical and Operative Surgery, Meath Hospital, and County Dublin Infirmary, &c. With a Memoir of the Author by ALEXANDER OGSTON, M.D., Regius Professor of Surgery, University of Aberdeen; Surgeon-in-Ordinary to the King in Scotland. Pp. 484. 25 plates and 52 figures. London: Baillière, Tindall, and Cox. Price 10s. net.

AT the time when Sir William Stokes undertook to give his services as a Consultant Surgeon to the troops in South Africa, he was engaged in revising for publication several articles which appear in this volume. The task of editing the remainder was entrusted by Lady Stokes to Mr. William Taylor, who had been associated with her husband for many years. It is well that such a book should be issued. The exact position of any man known in common parlance as "great" can only be finally determined when sufficient time has elapsed to allow his life's work to be thoroughly tested and approved. The personality of the man may either bring undue favour or unmerited discredit upon the work which he has done, and hence it is a good thing to have published in one volume the more important papers which have issued from his pen. The memoir of the author is a worthy introduction written by an almost lifelong friend, and gives one a clear intimation of the manner of man Stokes was. Born of a medical family of no little renown, he was brought up in the midst of professional surroundings, and doubtless early obtained a bias in favour of things surgical. Carrying all before him in his student days, he migrated to Vienna, Berlin, and Paris in turn, and took the opportunity of studying under such men as Jaeger, Langenbeck (the younger), Skoda, Von Graefe, Virchow, and many another celebrity. He was early appointed Surgeon to the Meath Hospital, and thenceforward devoted himself to surgical work, bringing to it a mind trained in the methodical German school, and a clear keen intellect which enabled him to utilise the material progress made by others, adapting and modifying it as seemed best. The result was that the credit of the brilliant succession of Irish surgeons in Dublin was, if anything, enhanced at his hands, and that Stokes of Dublin stands for the best traditions of Irish surgery. In this volume are collected together most of the papers dealing with subjects in which Stokes was particularly interested. The first deals with aneurysm, and contains a full account of his well-known case of ligature of the Abdominal Aorta undertaken in 1869. In this connection it is of interest to note that Stokes had definite doubts as to the real value of Compression in the treatment of Aneurysms—that treatment so belauded by the Irish surgeons of an earlier generation—recommending that in the so-called "surgical" aneurysm the treatment by pressure should not be unduly protracted. For traumatic aneurysm excision of the tumour with ligature of the proximal and distal vessels was by him considered to give the best results. Rhinoplasty, goitre, various types of amputation of the thigh, excision of joints, and many other subjects are dealt with in this work, and one has no hesitation in saying that, whatever a surgeon's ability, he will find much in this book which will refresh and stimulate him. Stokes had a wonderfully graphic pen, and we can well recommend this work to the notice of those who would not only strive for good results, but also endeavour to follow in the steps of those who could write well, clearly, and with interest, of their operations, as well as do them well. Too frequently the excellence of the work of British surgeons is discounted on the Continent from their lack of energy in writing it up; the publication of books such as that of Stokes will do something to remove such an uncalled-for depreciation.

Die Impfstoffe und Sera (Vaccines and Serums). L. DEUTSCH und C. FEISTMANTEL. Leipzig: Georg Thieme. Pp. viii + 285. 8vo. Mk. 6.

THIS useful and in some respects unique book will be of help not only to those who take an intelligent interest in the newer developments—both practical and theoretical, human and animal—of rational therapeutics, but also to those actually engaged in the preparation of sera, vaccines, and similar products.

The first portion, chiefly theoretical, deals with the various phenomena bearing on immunity and gives concise accounts of vaccination, virulence, protection, methods of acquiring protection, &c. So far as possible historical order is followed; the various theories of immunity with all that pertains to them are described and discussed. At the end of the first part there is a comprehensive list of the literature on the subject.

The second, more practical portion, describes in detail the methods of preparation of various vaccines and the effect of vaccination not only against small-pox, but also against plague, typhoid, cholera, hydrophobia, &c., and in animals against anthrax and various other diseases, in a manner showing actual acquaintance with the subject by the writers. Another distinguishing feature of the book is the description of the modes of packing and labelling the sera prepared by various institutions and manufacturers, together with the technique of their preparation in considerable detail, while the clinical indications for, and the symptoms following on, their administration are also set forth.

Among the more notable omissions and errors we remark that nothing is said about Koch's latest tuberculin-preparation (Bacilli-emulsion), while the curative use of tuberculin is also kept too much in the background. No mention is made of "Coley's fluid" for the treatment of malignant tumours. Under *Typhoid* we find the highly misleading statement that it is the difficulty of *distinguishing* B. typhi abd. from B. coli and its congeners which makes the detection of B. typhi abd. difficult in suspected water. This reveals a lack of acquaintance with modern methods. Chantemesse's results of a year's work with his anti-typhoid serum are also not quoted. The article on snake-venom is weak, and no mention is made of the important practical point of its rapid deterioration in warm climates.

An index would be a useful addition to a book of much interest and having many commendable features.

A Manual of Medicine. Edited by W. H. ALLCHIN, M.D. (Lond.), F.R.C.P., F.R.S. (Edin.); Senior Physician and Lecturer on Clinical Medicine, Westminster Hospital, &c. Vol. IV. "Diseases of the Respiratory and of the Circulatory Systems." London: Macmillan & Co., Ltd. New York: The Macmillan Co. Pp. 488. 32 illustrations and 3 coloured plates. Price 7s. 6d.

THIS, the fourth volume of the series, is perhaps less remarkable than its predecessors. The advance in the pathology and symptomatology of the specific and general diseases, and of the diseases of the nervous system, has, in the last few years, been so great that writers on these subjects have had abundant more or less new material to deal with, but with respect to diseases of the thoracic organs the advance has been less marked, so that the authors, in this volume, have had to proceed along the old lines. Still, this book as a whole contains a full and well-written account of the diseases of the pleura and lungs, of the pericardium and heart, and of the large vessels. In particular, the physiology of respiration is very well done, and contains exactly what is wanted by those who studied physiology many years ago. The article on the physical signs of the diseases of the lungs is also much to be commended, for it is comprehensive and yet avoids the over-elaboration which is so fashionable. Another excellent thing about the book is that, for the most part, the details of treatment are given with great care and fulness, the dose of the drug being specified and the method of administration, if all out of the way, being described. There are, however, one or two methods of treatment about which more should have been said. For instance, the open-air treatment of phthisis is, of course, mentioned, but in a book of this kind the student or practitioner would be entitled to find a full account of the rigours of Nordrach and of the curious effects of exercise on the temperature-chart and night-sweats which are observed in patients undergoing treatment there. Another method which should have been fully described is that of the treatment of cardiac affections by baths and exercises as it is carried out at Nauheim.

We think that the book suffers somewhat from the arrangement of the matter, especially in the wide separation of the pathology from the symptoms. In so many of the affections of the lungs the pathological changes reveal themselves so rapidly by changes in the physical signs that the two should be described almost side by side. The arrangement has also entailed a great deal of repetition on the part of the writers; and so marked is this in the section on the diseases of the arteries that the reading of it becomes wearisome in the extreme. Another fault in the book is the insufficiency of the index. This we pointed out when reviewing one of the earlier volumes, and as the number of the volumes increases the necessity for a full index becomes greater.

At the conclusion of the volume there is a learned article on œdema by the editor, which brings the pathology of that condition up to date.

Matière Médicale Zoologique. Histoire des Drogues d'Origine Animale. Par H. BEAUREGARD. Revisé par M. COUTIÈRE, avec Préface de M. D'ARSONVAL. Pages xxxi + 396. 144 woodcuts and photographs in the text, and 4 coloured plates. Paris: C. Naud. Price 12 francs.

ALTHOUGH Dr. Beauregard did not live to see the publication of his book, he was permitted to correct its proof-sheets upon his death-bed. He seems to have had a *penchant* for the study of comparative zoology, and he turned it to practical account by applying it to the animal materia medica. He did not, however, follow this line of research merely because he possessed an abstract love of this branch of science; but, also, because he saw that it would bestow a new interest and shed a new light upon the subject, and thus create a greater attraction for it in the minds of his students. We can well believe, in the words of M. D'Arsonval, that "sa vie peut se résumer en deux mots: Amour du travail; amour de ses semblables." The vegetable materia medica in its botanical, chemical, and microscopical aspects now receives adequate attention. Animal materia medica, notwithstanding the recent development of organotherapy, is, with the exception of very few articles, neglected. By bringing to it the anatomical and histological method of Beauregard many chemical and biological problems will be answered, even as many have been answered by the adoption of the same methods of research in the field of vegetable pharmacognosy. This volume is the direct outcome of Beauregard's researches. In fact it contains many of the results of his work which have not hitherto been published. In so far as it deals with the anatomical and histological characteristics of animals yielding drugs it is unique.

Some of the sections, notably those treating of musk, castor, civet, and the vesicating insects, are worthy of the designation "monograph." The one on musk occupies 32 pages, and the one dealing with vesicating insects over 50 pages. A considerable number of almost unknown drugs find a place in this volume, such as hyraceum, ægagropila, bezoar, the skink, vipers, ray-liver oil, cray-fish, trehala, and axine.

The chemistry of the various drugs has not been altogether neglected by the author, but it is far from satisfactory; for instance, there is no indication given as to the chemical composition of lanoline, and the obtaining of pepsin is dealt with in a summary fashion. We think it a matter for regret that the researches of English workers have been so largely overlooked, especially as it has resulted in the exclusion of any information concerning the thyroid body.

The drawings, photographs, and plates, together with the excellent letterpress, combine to make this an interesting as well as an instructive addition to medical literature.

Serum-Therapy, Bacterial Therapeutics, and Vaccines. By R. T. HEWLETT, M.D., M.R.C.P., D.P.H., Professor of General Pathology and Bacteriology, King's College, London, &c. Pp. 262. London: J. and A. Churchill. Price 5s. nett.

WE are glad to welcome an English book on the important subject of serum-treatment and vaccines, which is emphatically the treatment of the future. Professor Hewlett's little book gives a commendable account of the present state of our knowledge of immunity and kindred subjects, and discusses the manufacture and standardisation of serums and the practical application in disease. The account of the laboratory methods involved is very full and valuable. The practical side is, perhaps, less strong, but it must be remembered that at present serum-treatment is still largely in an experimental stage, and definite advice is difficult to give on this subject. The book is up-to-date in its information, well got-up, and excellently printed.

A Manual of Toxicology. By ALBERT H. BRUNDAGE, A.M., M.D. 2nd edition. Pp. viii + 375. London: Baillière, Tindall, and Cox. 6s. nett.

THIS manual was written to provide a ready means of review of the salient points of toxicology and was prepared, in the first instance, for the author's own students. Before proceeding to consider individual poisons a section is devoted to definitions, classifications, and general directions, that is to say, a general survey of the subject given in a very succinct form. In dealing with separate poisons the maximum and fatal doses, together with a few general facts, are first given, then the symptoms leading on to the particulars of treatment. In Part III. the symptoms are classified, and the poisons which they indicate are grouped under them. Then follow other "parts" treating of the identification of, and search for, poisons, the signs and modes of death, the interpretation and treatment of coma, chronic poisoning, and a miscellaneous collection of tables among which we find a pulse table, income and expenditures of life, weights and measures, temperature equivalents, weights and measures of viscera, and others quite as germane, and not more so, to toxicology. As a reference book for emergencies it will no doubt prove useful.

St. Bartholomew's Hospital Reports. Edited by NORMAN MOORE, M.D., and D'ARCY POWER. Vol. XXXVIII. Pp. 258 and 206. London: Smith, Elder and Co. 1903.

AS usual these well-known reports contain a number of interesting papers by members of the staff and old students in addition to the more statistical reports. Of the former there are fourteen, of which six, or nearly half, are contributed by active members of the medical staff. Dr. Gee, who is a most faithful supporter of these reports, writes on hæmatemesis in a series of *obiter dicta* which recall his well-known (and lately published) aphorisms which originally appeared in these reports. Mr. Power describes a number of intestinal cases, and discusses their surgical treatment. The diagnostic value of melanuria, or presence of the pigment of melanotic growths in the urine, is critically dealt with by Dr. Garrod in a very valuable essay. Dr. Herringham's contribution on purpura, especially in sarcoma, lymphadenoma, and tuberculosis, recalls, in its economical respect for words, the writing of his senior colleague, Dr. Gee. The treatment of Tinea Favosa by Izal is shown by Sir Dyce Duckworth to be extremely successful. Mr. Jessop's paper on the Pathology and Prognosis of Glioma of the Retina contains many important points: the growth he regards as an endothelioma; if it recurs it does so with such rapidity that a patient who is alive one year after removal may be considered safe. There are a number of other interesting papers by Drs. Thursfield, Forbes, Inchley, Pryce, and Campbell, which cannot be further referred to.

Saunders' Year Book of Medicine and Surgery. 1903. 2 vols. Pp. 691 and 670. London and Philadelphia: W. B. Saunders and Co. Price 13s. nett per volume.

THESE two volumes form a well-got-up and well-illustrated book of reference to the principal advances made in the fields of medicine and surgery in the past twelve months. The first volume contains Medicine, Pathology, Bacteriology, and Hygiene; the second Surgery Obstetrics, Gynæcology, and special departments (Ear, Throat, &c.). The work seems to have been well done throughout, and we can safely recommend it to those who are in need of an epitome of recent work, or who wish for an easy way of keeping up with the ever-increasing flood of medical literature.

The Scottish Medical and Surgical Journal. Edited by NORMAN WALKER, M.D., F.R.C.P. Ed., and HAROLD J. STILES, M.B., F.R.C.S. Ed. Vol. XI. Edinburgh: The Scottish Medical and Surgical Journal, Limited.

THE last volume of this periodical contains many papers of interest. Among them we may mention the contribution by Dr. James Ritchie and that by Dr. John Thomson on the subject of Pyelitis in Infants, a condition which is now becoming well recognised; by Mr. F. M. Caird on Gastro-jejunostomy; by Dr. F. Gardiner on the X-rays as a diagnostic agent in Phthisis; and by Dr. R. A. Fleming on Regeneration of Nerves. There are also valuable epitomes of recent work in many departments of medicine and surgery, and much instructive editorial comment.

A Manual of Practical Medical Electricity. The Röntgen Rays and Finsen Light. By DAWSON TURNER. Pp. xx + 396. 168 illustrations. Demy 8vo. London: Baillière, Tindall, and Cox. Price 7s. 6d. nett.

WE congratulate Dr. Dawson Turner on the appearance of a third edition of his book, which is thoroughly revised and brought up to date. Nearly seventy pages have been added on treatment by Röntgen Rays, by the Finsen lamp, and by ultra-violet light. It is refreshing to find evidences of original work in a book on medical electricity, for only too often these books present a wearisome sameness, and show many signs of being compiled with the aid of scissors and paste. Dr. Dawson Turner's account of his study of the ultra-violet radiations yielded by the different lamps in use at the present time is a good piece of work, which will be of great use to those who are following up the same line of treatment. The condenser spark-lamp with iron terminals appears to hold its ground as the best source of ultra-violet radiations, but it must not be forgotten that we do not yet know whether the rays which act best upon lupus belong to the blue, the violet, or the ultra-violet regions of the spectrum.

The author does not touch upon the strange influence of the static brush-discharge upon cutaneous disorders; a line of work which MacIntyre of Glasgow has brought to the notice of the profession, and one which certainly has proved to be useful in lupus, in chronic ulceration, and in some forms of superficial epithelioma.

The subject of high-frequency treatment is referred to, and its principles are described, but the complicated technique recently introduced into this mode of treatment is (shall we say, rightly) ignored. High frequency is a form of electrical application which has yet to find its proper position; at present it labours under the disadvantage of having fallen into the hands of unqualified persons, and of being recommended for all sorts of quite unsuitable cases, so that its general influence upon the progress of electro-therapeutics threatens to be a bad one.

Electricity in Medicine and Surgery, including the X-ray. By WILLIAM HARVEY KING. New York: Boericke and Runyon Company. Pp. 296. 116 illustrations. Second edition.

THIS volume touches upon most of the matters usually dealt with in books on medical electricity. It is written in a diffuse and slipshod style which fails to impress the reader favourably. Many of the sections, even among those which deal with subjects of prime importance from the point of view of electro-therapeutics, are badly done, and give a poor impression of the present state of our knowledge concerning them.

Die Ætiologie des Akuten Gelenkrheumatismus. (The Ætiology of Acute Articular Rheumatism.) By A. MENZER Stabsarzt, &c. With a Preface by Prof. Senator, and 5 plates. Berlin: Aug. Hirchwald.

THIS little book has appeared at a time in the history of rheumatism when it cannot fail to be of interest to the reader; and although we may not be prepared to follow Dr. Menzer in all his statements, we must recognise his boldness of view and the value of the suggestions which he throws out. The author's conception of the disease is essentially that of an infective process. It is a "symptom-complex," the expression of a blood-infection which has penetrated the body from without. The entrance of the infection is made from the region of the palate, tonsils, and upper air-passages. Dr. Menzer is of opinion that there is no specific bacterial cause; but, nevertheless, we find him admitting that strepto-diplococci are by far the most frequent excitants of the disease. Here, as in other of his writings upon this subject, he wavers between the specific and non-specific views, using each one in turn to assist him when he is in difficulties. Especially interesting are the investigations of the author upon angina faucium, and his remarks upon this subject. Dr. Menzer lays much stress on the probable value of an anti-streptococcic serum, prepared by the method of Tavel and Krunbein, and he treats with a healthy scepticism the specific action of the salicyl compounds. For our part we regret that he should consider acute articular rheumatism as a disease in itself, though it must be admitted that this opinion is widely maintained in Germany. The book is illustrated by five plates, and there is a useful bibliography. No one interested in the study of rheumatism can fail to be impressed by this book, which is a most suggestive one.

Death and Sudden Death. By P. BROUARDEL, Professor of Medical Jurisprudence, Dean of the Faculty of Medicine, Paris; and F. LUCAS BENHAM, M.D., B.S. Second edition, London: Baillière, Tindall, and Cox. Price 10s. 6d.

DEATH and sudden death are subjects which concern every member of the medical profession. Sudden death from unexpected or obscure causes may lead to legal proceedings, the results of which must often depend largely upon medical evidence. For these reasons, as well as for the pathological interest which such cases have, the subject is well worth studying. Professor Brouardel's book, which has been translated by Dr. Benham, is an excellent volume, and deals fully with the various medico-legal questions that may arise. The first part of the book deals with the signs of death, rigor mortis and putrefaction. The uncertainties of many signs are pointed out and their relative values noted. In summing up, Professor Brouardel says: "A physician need not often hesitate over the diagnosis of death; but he must know how to make this diagnosis just as he knows how to make that of typhoid fever or meningitis. Some cases present the condition of apparent death, which may be more or less prolonged. The physician alone is capable of making the diagnosis in these; everyone else is incompetent." The second part of the book consists of a series of lectures on the causes of sudden death, and those occurring in connection with the different systems are taken in turn. Among the many interesting chapters in this part, that dealing with death from inhibition may be mentioned. Here the author shows how insignificant blows upon various parts of the body may be followed by death, which the medical man may find very difficult to explain. From beginning to end the book is very interesting and very instructive, and can be most cordially recommended to the notice of all professional readers.

Elements of Pharmacy, Materia Medica, and Therapeutics. By WILLIAM WHITLA, M.A., M.D. Eighth edition. Pp. 632. With woodcuts. London: Henry Renshaw.

THE new edition of Whitla's *Materia Medica* is practically a new book. It has not only undergone thorough revision, but the size of the page and of the type have been increased, so that now it is uniform in appearance with the author's *Dictionary of Treatment.* The section dealing with non-official remedies is almost entirely new; it consists of about a hundred pages, and comprises over five hundred drugs, concerning many of which it is often difficult to obtain information. For instance, on one page we find the following items: — Castanea, caullophyllin, cedron, cerasin, ceresin, cetrarin, chaulmnoogra oil, and chekau. It is an excellent work of reference, not only as regards rare remedies, but also for practical notes upon therapeutics. We think that it would be an improvement if the part treating exclusively of chemical reactions were incorporated with the official remedies. Dr. Whitla in writing of the glycerophosphates has evidently overlooked the fact that the tonic action of the compound syrups depends in no small measure upon the strychnine they contain. On page 142 there is some confusion between the natural orders *Loganiaceæ* and *Apocynaceæ*, as there is also between gingerin and gingerol on page 290.

The popularity of this work as a student's manual is thoroughly justified, and its improved form will make it still more acceptable. As a pithy, compact work of reference for the general practitioner it would be difficult to excel.

THE PRACTITIONER.

NOVEMBER 1903.

A CLINICAL LECTURE ON EXTRA-UTERINE GESTATION.[1]

BY SIR HALLIDAY CROOM. M.D., P.R.C.S.E, &c.,
Consulting Gynæcologist, Royal Infirmary, Edinburgh, &c.

GENTLEMEN,

I HAVE chosen the subject of extra-uterine pregnancy as that upon which to address you this afternoon for various reasons. First of all because the subject is one of the best landmarks in the development of surgical gynæcology. You know that this matter is a triumph of surgery. It is a trite and perhaps exaggerated saying that gynæcology has for ever passed away from off the shoals and quicksands of uncertain medicine to the surer waters of surgery. Be that as it may, the fact remains that until extra-uterine pregnancy departed from the hands of the physician to those of the surgeon it made practically no progress at all. Previously the condition had been regarded only as a curiosity. No doubt extra-uterine pregnancy has been known for centuries; in the year 1876 Parry wrote his famous book giving 500 cases of extra-uterine pregnancy, but it was not until Tait in 1883 took the subject away from medicine and made it a province of surgery that the matter was put upon a definite and progressive basis. My second reason for choosing this subject is, because I think that, to the general practitioner, it is a subject which is not properly focussed. Lastly, I have chosen it because the school to which I have the honour to belong has performed a fair share of work in elucidating this matter.

In the first place, before we reach the subject, it is necessary that you should consider for a moment the way in which extra-uterine gestation is classified. If I can make this sufficiently clear to you, I feel that I shall have done what most of us want to-night. I hope to reduce this condition of extra-uterine pregnancy to its very simplest elements ; to wipe away—for good, if I can—all the dust that hangs about it and bring it down to the simplest terms. If you look at the books, you will see that there are any number of extra-uterine pregnancies described. First of all you will find described "abdominal pregnancy," that is to say, primary abdominal pregnancy. With regard to secondary abdominal pregnancy, nobody doubts that; no one questions that a tubal pregnancy may rupture into the abdominal cavity. But is there such a thing as primary abdominal pregnancy ? I believe there is not. It is, of course, possible to conceive that a spermatozoön may pass out of the fimbriated extremity of the Fallopian tube and meet the ovum in the abdominal cavity, where the latter may become impregnated; but I think this is most unlikely. One can place no limits to the possibilities of an enterprising spermatozoön ; but, to say the least of it, such a coincidence is very problematical. Only one case of the kind has been described—that of Schlectendahl—and I regard it as wanting confirmation. Therefore, I think, we may at once get rid of primary abdominal pregnancy, and may regard it as of no interest, at least to the general practitioner.

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[1] Delivered at the Medical Graduates' College and Polyclinic.

But is there such a thing as "ovarian pregnancy" ? Leopold and Martin and Patenko have described cases; that is to say, they have described cases in which they have removed tumours of the ovary of long standing, which contained either a lithopædion or fœtal bones. But as such cases are extremely rare, we may for all practical purposes dismiss them. How, indeed, are you to explain ovarian pregnancy in such cases, if they exist ? Only by imagining that the spermatozoön enters the Graaffian follicle after its rupture and before the escape of the ovum. Indeed there might be a follicle with two ova, one escaping and one remaining, the latter being impregnated. This is, however, mere theory, and we may put ovarian and abdominal pregnancy out of the question altogether; they are curiosities which do not interest the general practitioner in any degree. What we are contented to have is classic cases—those which you are likely to meet in your practice. Therefore, it comes about that for all practical purposes we have only to deal with "tubal pregnancy" in detail; and in the limits of this lecture it will be impossible for me to go beyond early tubal pregnancy.

The Fallopian tube can be divided into three parts, and the varieties of tubal pregnancy are named according to what parts they occur in. There is the *interstitial* form, which occurs in the uterine substance; the *infundibular*, which is situated in the fimbriated extremity; and the *ampullar*, which is in the central portion between the two.

The first question at issue comes to be, how does tubal pregnancy ever come about at all? How is it possible for a woman to develop an ovum in this situation? That brings me back to another question, namely, Where does impregnation take place? Does the ovum meet its fate in the Fallopian tube or in the uterine cavity? All the older teachers and most of the present ones believe that impregnation takes place in the tube; while others, such as Tait, who is as usual emphatic, says that it never takes place anywhere except in the uterine cavity in normal cases. The question is undecided, and it must remain so, because we cannot get any good proofs in the living human being; all our knowledge is taken from the lower animals. So we must take it in the meantime that it occurs in the tube. Then, before we can go further, the second question arises:—If it takes place in the tube, or even if it occurs in the uterine cavity, by what mechanism is the condition produced? How does a woman get a tubal pregnancy at all? Of all the theories which have been advanced, perhaps none have been more beautiful — though unfortunately it is not true — than that advanced by Lawson Tait. He says—what is absolutely true—that most extra-uterine pregnancies occur in women who have had some form of salpingo-oöphoritis, and as far as my limited experience goes, all, except one, of the cases upon which I have operated for this condition have had such trouble. Tait says that this trouble leads to the condition which we are discussing by producing a desquamative salpingitis. Suppose you have a normal condition of things, with the cilia in the uterine cavity working upwards towards the tube, and the cilia in the tube working downwards towards the uterus. Then the latter cilia push the ovum down, while those in the uterus push the spermatozoa upwards. Ovum and spermatozoön meet in the uterine cavity and there develop. But what occurs when there is a condition of desquamative salpingitis? In desquamative salpingitis the whole of the cilia in the tube have disappeared; and therefore you will observe the little ovum enters the tube, but, from want of cilia, it can get no further. Meanwhile the cilia are working upwards all the same in the uterine cavity, and are helping the spermatozoa to ascend. Thus a spermatozoön, you will observe, may get in along the Fallopian tube, where there is nothing to oppose its entrance, and may meet its fate there with the little ovum which has remained in the tube. That is Tait's idea, and you will be able to appreciate that it is a pretty theory. It has, however, recently been proved that the uterine cilia do not work upwards, so that Tait's ingeniously-simple theory cannot be accepted. Then, gentlemen, if it be *not* from any desquamative salpingitis, the occurrence may be brought about by a kinking in the tube, by a thickening in the mucous membrane, by a change in the tube, or by a congenital deformity. At all events, it remains a fact, in whatever way you explain it, that previous to the development of extra-uterine pregnancy a woman mostly, if not always, has had some affection of her appendages.

We now come back to the classification of the varieties of tubal pregnancy. Let us reduce tubal gestation to its simplest terms. First of all, there is the interstitial form, namely, that in which the ovum develops in the actual uterine structure. There the ovum develops, actually within the wall of the uterus itself, within that portion of the tube which is contained in the wall, and is therefore not really extra-uterine. It is possible that there may be two varieties of this form—primary interstitial pregnancy, or secondary interstitial pregnancy, caused by the invasion of the uterine wall by a growing tubal pregnancy. Most, if not all, these cases are cornual pregnancies. The environments of these gestation-sacs differ from those of the ampullar and infundibular forms: the muscular fibres hypertrophy and rupture is inevitable. This usually takes place about the fifth month, and is uniformly fatal. Therefore, though it may be a profoundly interesting variety, it is seldom, if ever, recognised before the fatal occurrence takes place, and all I venture to say to you is, that should it be your good fortune to meet and recognise such a case, the sooner you interfere the better. Therefore I think we may put the interstitial variety out of court.

What about the infundibular form? That is another question. The ostium is open, and if by any accident an ovum should, for want of cilia, remain fixed at its distal end, in the tube, there it will develop, for want of progressive power downwards. What would be the result? It is a result which you or I cannot help or avoid. It simply means that the little ovum, loosely attached and developing where the tube is open and exposed, in whole or in part, drops into the peritoneal cavity, where it is absorbed. Therefore you have what is known as "tubal abortion." It would be wrong to say that that means nothing. It often means a good deal, because associated with this you often have very considerable hæmorrhage. But sometimes you have a little ovum remaining near the infundibular end and developing there; but for some reason or other the energy of growth ceases and the ovum remains fixed in the shape of a mole. Such moles I have met with on two occasions and lifted them out of the pelvis, where they had no adhesions: they were simply sacs containing old blood-clot. At the same time it must be kept in view that such a pregnancy may have the same life-history as an ampullar pregnancy, which we now proceed to discuss, for the practitioner is not likely to recognise a mole till it is taken out, or a tubal abortion till it is over.

We come then to the third variety—the ampullar—and it is to this that I want to direct your special attention, as it is the form which claims the interest of the practitioner of medicine; because, whatever other teachers may say on the matter, it is a certain fact that an ampullar pregnancy can be readily diagnosed before rupture, and if that can be done, you greatly diminish the risk of extra-uterine pregnancy. Although it may seem to you a strong way of putting it, I do not think there is any operation which is more satisfactory than that for extra-uterine pregnancy before rupture, although it may seem difficult on the face of it. You know what you are dealing with, and you can often get it out without rupture. If it does rupture, it does not matter. It may seem to many of you an operation which you do not care to tackle; but if once you diagnose it before rupture, there is no operation which is easier as long as the condition is not advanced.

I pass over anatomical questions such as the vascularity of the tube, the disappearance of the plica, and the replacing of the muscular tissue by fibrous tissue, and so on. That is interesting, but in a lecture like the present it is rather out of place.

The life-history of ampullar pregnancy is the side which is important to consider. What is the life-history, as a rule, of ampullar pregnancy? I have not troubled you with the pathological changes, but I will give you the clinical facts. As the ovum grows in this part of the tube, naturally the walls become thinned, and this thinning is the essential matter. What is the issue? There are two issues of which it is necessary to speak to-night. In a classical case the natural history of the condition leads to one of three results. First of all, there may be early rupture. Now, when I say early rupture, I mean rupture between the second and the third week, very early in pregnancy. When such a rupture takes place, of course the symptoms are comparatively very slight. A woman complains of pain in her side associated with an evanescent syncope, and after a short time she recovers, and a local examination will reveal a little effusion at one or other side of the uterus. It is said that she has got injured by lifting a weight. The last case I saw was that of a woman who had the rupture while getting out of a tram-car; at least, from her suppressed menstruation and various subjective symptoms. I assume that it was early extra-uterine pregnancy. These cases need not occasion any alarm, because the hæmorrhage is slight and is, sooner or later, absorbed. The worst that can happen is that they may develop into one of those moles which I have described. But the point is that, as a rule, these early ruptures do not occur, or if they do occur, they are not observed. Therefore I will not do more than point out to you that, when a woman has a pain in that region, low down in the pelvis, and when her menstruation is delayed, you have every right to expect early tubal pregnancy. But you have no right to do what some authors advise, to cut down and take it out. These early extra-uterine pregnancies do not give rise to trouble as a rule, and do not require operative treatment.

But far otherwise is it with the second possibility, namely, that it may go on developing and getting more vascular; that the placenta in growing may press on the walls of the tube, which tend to get thinner, the fibrous tissue relatively increasing while the muscular elements atrophy, until ultimately, late in early pregnancy—between the eighth and twelfth week— the woman has a sudden rupture of this tube, and probably dies. Between the eighth and twelfth weeks is the time when this condition can be recognised, and when it is best treated. It will generally come under your observation as practitioners at that time. Now let me try to point out to you two things. I have said that the tube ruptures between the eighth and twelfth week. The crux of the whole matter is, Where does it rupture? Whether the woman's life is immediately jeopardised or not, depends on the direction in which the rupture takes place. The broad ligament, as you know, is divided into two layers, between which there is cellular tissue. Suppose that the tube ruptures at such a point that the blood is extravasated into the free peritoneal cavity. There is no circumscription in the abdominal cavity; it can contain almost any quantity of effusion, and the woman rapidly bleeds to death, unless the case is operated upon promptly. Unless that is done these cases are promptly and uniformly fatal. It is therefore upon these cases that the practitioner requires advice; when the woman is blanched suddenly and there is pain at the side, that is the time for the practitioner either to operate himself or to get someone else to do so. It is then that the woman's life can be saved, and an hour may end the matter.

It is another matter when the tube ruptures the other way—that is to say, when the extravasation takes place between the layers of the broad ligament. Of course it is circumscribed then, and it has to stretch the layers of the broad ligament little by little. The consequence is that the hæmorrhage is gradual, and syncope less marked. But you must not run away with the idea that the two layers of the broad ligament are so powerful that they prevent bleeding, because they can be stretched to almost any extent, but the process is slow and gives the practitioner plenty of time to watch the case and to interfere.

According to the site of the placenta and the direction of the hæmorrhage, so will the fœtus follow. If the hæmorrhage be into the peritoneal cavity, the quantity may be enormous, and the fœtus will promptly follow the hæmorrhage into the cavity. If, on the other hand, the hæmorrhage be into the broad ligament, and the placenta be situated there, then hæmorrhage between the layers of the broad ligament will be less rapid, and the fœtus will follow the extravasation down through the layers of the broad ligament. I will mention a case bearing upon this :—

Mrs. M., aged 38, was a multipara. It was four and a half years since her last child. For two months she had had amenorrhœa, with subjective signs of pregnancy somewhat exaggerated, followed by irregular hæmorrhages, and passage of a decidual membrane entire. At the end of the third month severe pain and collapse occurred, pointing to internal hæmorrhage. Three weeks afterwards there were again severe pain and collapse (second internal hæmorrhage). A third internal hæmorrhage occurred a week later. Examination, after the third hæmorrhage, revealed a large pelvi-abdominal tumour, soft and elastic, extending half way up to the umbilicus on the left side, with no auscultatory sounds. Abdominal section was performed and a ruptured tubal pregnancy found. Removal of fœtus and placenta was effected, and the sac packed and drained. You will see from this case that sometimes the rupture of the sac takes place, but the broad ligament re-encysts the pregnancy; this second sac may then rupture.

In some cases, if the placenta goes first, you will find it in the sac by itself, or if the fœtus escape first, it will be found in a sac by itself. But the great point in the history of these extra-uterine pregnancies is the direction of the rupture, whether it be intra- or extra-peritoneal.

I may quote to you shortly a typical case of extra-uterine pregnancy before rupture :—Mrs. P., aged 27, was a primipara. There was a history of old pelvic inflammation. Subjective signs of pregnancy occurred, associated with two months' amenorrhœa, and followed by irregular hæmorrhages, periodic pain, and the passage of a decidual membrane. *Per vaginam*, there could be felt an elastic enlarging tumour on the left side and behind the uterus. The uterus was deflected to the right and slightly enlarged. Abdominal section was performed, and a three months' tubal gestation-sac removed entire.

We now come to the question "Can we diagnose the condition before rupture?" If I can succeed in making you believe this, I am contented. Aveling said it was fairly easy to diagnose extra-uterine pregnancy, while Tait told me once to my face that, if a man said that he could diagnose extra-uterine pregnancy before rupture, he would not believe him again either on that or any other matter. So, you see, opinions vary. Still I think I can prove that you can, with absolute certainty, diagnose extra-uterine pregnancy before rupture. Let us try to look shortly at the conditions available for diagnosis of extra-uterine tubal pregnancy of the ampullar form about the eighth or twelfth week before rupture. The following points I think you will find extremely important. First, of course, you always find more or less of the subjective signs of pregnancy, sometimes even exaggerated. It is unfortunate that the mammary signs are not of value, the majority of cases occurring in multiparæ; but it is an interesting thing to find a change in the mamma, perhaps to the extent of the development of milk. Second—and this, I think, every case which I have had, except one, proves up to the hilt—there is a long interval between extra-uterine pregnancy and the pregnancy which preceded it. It is seldom indeed that a woman has a living child and shortly afterwards is the subject of a tubal pregnancy. In all cases taken from case-books and text-books there is hardly one in which there has not been a long interval between the last child and the extra-uterine pregnancy. Another very important point is that there will often be some utero-ovarian disease. I have referred to what Lawson Tait said about salpingitis: there is an element of truth in it, in that there is some morbid condition of the uterus and appendages which predisposes to extra-uterine pregnancy. You can scarcely imagine an extra-uterine pregnancy taking place unless some change has occurred in the appendages since the previous pregnancy.

The next point is what is called "disordered menstruation." That, however, is a misnomer: it is not disordered menstruation at all. What I think you will get in the vast majority of cases is, that a woman will miss one month, and after that month will have irregular hæmorrhages every ten or fourteen days. There is first of all amenorrhœa, followed by irregular hæmorrhages. It is really this combination of amenorrhœa and irregular hæmorrhages which characterises this condition.

Another symptom, however, which is rarely wanting, is localised pelvic pain, which is not usually constant, but is more or less proxysmal and confined mainly to the side of the pelvis in which the gestation-sac lies. It is, no doubt, due to the rapid distension of the tube-wall.

Given a woman with those symptoms, you next search for information from an examination *per vaginam*. What will you find there? As to this I should like to be emphatic, because what I shall tell you now will clear the way for you immensely. After consideration I should say that the following are the characteristics of extra-uterine pregnancy in its most typical form, and if you find them you may rest assured that your course is clear. First of all you will find a tense cyst. It is difficult to find words which will express what one feels with the fingers. You will find a distinct swelling, a cystic swelling, on one or other side of the uterus. Furthermore you will find it sensitive. I have seen an extra-uterine pregnancy burst under the fingers of the man who was examining it, and the woman had an internal hæmorrhage on the spot: therefore, you must examine these conditions with the utmost care and gentleness. Nobody gets any information by poking the fingers into the woman's vagina and pressing. It is by gently touching, especially in extra-uterine pregnancy, that you get all the information you want. I may mention, while speaking of this examination, that it is of no use unless the patient is put under deep anæsthesia, because there is always the resistance of the abdominal muscles, which the patient keeps tense. If you do not anæsthetise the patient, you may miss very important points in the case. Again, this tumour is not only cystic and sensitive, but it is pulsating. You would scarcely expect it to be otherwise seeing the amount of vascular development there is.

I have said that it is fixed. Now, its position is sometimes a matter of difficulty. One would naturally suppose that a tubal pregnancy so situated would be on one or the other side of the uterus. So it very often is; but do not be surprised if you find it behind the uterus, because, before the pregnancy develops, the tube may sink down and lie in the central sac of the peritoneum. Usually it lies on one side and a little to the back.

You have then obtained the history from the woman; you have examined the condition, and you have found a soft, tense, pulsating tumour fixed on one side. But that is not sufficient; you must go further and examine the uterus, and you will then find that it has shifted to the other side, to one side or the other. Here I would interpolate a single word. When the diagnosis comes to be between what is called hæmatocele and extra-uterine pregnancy, you will find that the position of the uterus will to some extent help you, because hæmatoceles are very rare, and they are mostly caused by extra-uterine pregnancy. In those which are not so caused, one of the most interesting facts is that the uterus gets pushed up, fluid gets below it and pushes it towards the brim of the pelvis. That is one of the diagnostic points. But the point for you to notice next is that you will find the os uteri almost always patulous and the body enlarged. With regard to this condition of the uterus, I may remind you that the uterus enlarges along with the extra-uterine pregnancy *pari passu*. As the extra-uterine pregnancy goes on, so does the uterus enlarge. Now, it only goes a certain length. Suppose the extra-uterine pregnancy were to develop on to the sixth month, then you would find the uterus not a bit larger than at the third month. It stops growing. Why should the uterus stop growing at the third month, and yet the pregnancy develop further and further? Because it is following Nature. You know that decidua and reflexa coalesce before the third month, and at the end of the third month they are very thick, soft, vascular bodies. But from that time onwards the mucosa gets thinner and thinner, until at full term the condition of the mucosa is simply that of a fibrillary membrane covering the outside of the chorion; and therefore the uterus stops developing just when this membrane ceases growing, at the end of the third month. Curiously enough the expulsion of this decidua is one of the most striking signs, if not the pathognomic sign, of extra-uterine pregnancy.

Sometimes the decidua redevelops. It is shed off at the third month, but it redevelops sometimes, and at term you find that this uterine cavity corresponds to a three months' pregnancy, and is covered with thick soft decidua. I had one of the most interesting experiences in this respect which has fallen to the lot of an operator. In operating on a case at full term, I found at the side of the extra-uterine pregnancy a uterus about as big as it should be at the third month. We make it a rule, if we can avoid it, not to operate in these cases until the full term. Nature asserted itself there, and before the operation took place this uterus went into regular labour, with regular pains; and on putting one's finger into the uterus one felt it contract upon it and the whole decidua was shed out entirely. Sometimes it occurs, therefore, that the decidua redevelops and is shed off at full term. If it should be your good or your bad fortune to get an extra-uterine labour at term, wait until the decidua is expelled, and then do your operation.

The expulsion of the decidua then is one of the most important factors in diagnosing extra-uterine pregnancy. It requires to be watched carefully. It may be expelled whole, but it is often expelled in part; but wait until you get it, unless you find that rupture is absolutely imminent. If you pay sufficient attention to this point you will have no difficulty in diagnosing the condition.

I will now refer to the symptoms which occur if rupture does take place. Suppose rupture to take place into the peritoneal cavity. How do you know it? The symptoms will be acute and sudden; there is always agonising pain; there will be the indications of internal hæmorrhage, presenting the usual features—shock, syncope and death. Supposing you are put face to face with a woman who is suddenly collapsed with internal hæmorrhage, the question arises, whether you should at once open the abdomen. On the whole I should say "yes," because most of these cases are due to extra-uterine pregnancy. At the same time you must remember that a hæmatocele may occur from causes other than an extra-uterine pregnancy. It has twice been my lot to open the abdomen distended with blood—where the women were unconscious—and to find no cause for the hæmorrhage whatever; but, although women with hæmatocele frequently recover, yet, in any case of doubt, there can be no reason why the abdomen should not be opened.

Here let me mention a case which shows the rapid absorption of a hæmatocele. Not very long ago a lady sickened on her honeymoon. Suddenly in the middle of the night she cried out that she was in great pain. She was collapsed and became unconscious. I saw her shortly afterwards and I had nothing to operate with. It was the middle of the night, and I was a long way from home. I therefore temporised in the best way I could, though the woman's abdomen was full of fluid blood, as I could tell by palpation. But she resumed her journey within a month, so rapidly does blood get absorbed in the abdominal cavity.

If the rupture be between the layers of the broad ligament, there can be no question that the symptoms will be less marked. The pangs of pain will be more transitory. Of course the hæmorrhage is circumscribed and the syncope is comparatively evanescent. Indeed the diagnosis between the two forms depends more upon the symptoms than the signs, and relatively more upon their severity. It must, however, be borne in mind that quite a large number of tubal pregnancies do not actually rupture, but the hæmorrhage, often times very great, is from the distal end of the tube, the ampulla remaining entire. And this, of course, renders the removal of the entire mass easier than it otherwise would be.

Now, as regards the treatment. Operative interference may be overdone in this as in many other tubal conditions. In very early cases after rupture, operation is not necessary. But there can be no question that, after the condition has been diagnosed, the sooner the gestation-sac is removed the better; and after rupture, where life is threatened, it is essential.

I need not here detain you by referring to the older methods, the injection of morphia and so forth, which have quite fallen into desuetude. Nor need I refer to a differential diagnosis between extra-uterine pregnancy and other affections of the Fallopian tube, because in any case removal is the obvious treatment. With regard to hæmatosalpinx, which is often difficult to diagnose from an extra-uterine pregnancy, it is really a moot question whether most of these so-called cases of hæmatosalpinx are not really extra-uterine pregnancies.

In what I have said to-night I have taken scrupulous care to deal with classic forms. I have led the way to no by-paths and have made no excursions into unusual conditions of extra-uterine pregnancies. With regard to those cases which re-encyst, of which the first case I quoted was an example, that is another matter. I do not think these appeal to the ordinary practitioner, though they may be interesting enough to those of us who devote themselves to this specialty. Still less this evening have I attempted to speak of full-term pregnancies. If I have succeeded in impressing upon you the means of diagnosing this condition before rupture, and the necessity of dealing with it at once, I have done all that I could hope to do in one lecture.

THE PRESENT POSITION OF OUR KNOWLEDGE WITH REGARD TO THE TREATMENT OF UTERINE FIBROIDS.

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THE advance made in scientific gynæcology since ovariotomy became an established operation must be reckoned as one of the most satisfactory features in medical progress during the last quarter of the nineteenth century. In order to be convinced of the magnitude of this advance we need only compare the recent discussion on deciduoma malignum at the Obstetrical Society of London with the debates on other subjects preserved in the earlier volumes of that Society's *Transactions.* The development of abdominal surgery beyond the limits of ovariotomy is closely linked with the evolution of antiseptic and aseptic surgery. The treatment of fibroids has been deeply influenced by this development. For several years its progress was relatively slow, because operations on the kidney and gall-bladder proved easier and less perilous than hysterectomy for fibroid as then practised. Since 1890, however, technical improvements and increased experience have rendered hysterectomy far less dangerous to the patient's life. In consequence wholesale extirpation of fibroids is practised at the present day, as the pages of the medical press show week by week. A theory has been advanced that all fibroids should be removed. This theory is stoutly contested by men of experience. The truth lies between the two opposed opinions, and the discussion of these opinions may conveniently form the basis of this contribution, as it will throw some light on the present position of our knowledge with regard to the treatment of uterine fibroids. The evidence gained by the results favourable or otherwise of operative treatment has proved most instructive to medical science.

A fibroid demands no immediate treatment when its sole symptom is its own existence. Champneys' statistics show how many women live and die with dormant fibroids. One of the boldest of operators, Murdoch Cameron, maintains that in his own experience not more than one third of the cases seen require surgical interference.[1] By "cases seen" he implies that many are not seen, the tumour remaining undiscovered. Howard Kelly, another renowned operator, emphatically stated at the recent Congress in Madrid that the man who maintained that every fibroid should be removed was greatly mistaken. When a fibroid of small size is detected accidentally, and it is clear that the patient's malady has nothing to do with the tumour, its removal is not justifiable.

The course of a fibroid, however, is extremely uncertain. We can safely prophesy what will happen if an ovarian cyst or a cancerous cervix be left alone, whilst even the most experienced amongst us can never feel sure that a fibroid hitherto symptomless will or will not remain quiescent. I strongly support the opinion that it is not right to urge an operation, when no symptoms are present, simply to secure the patient against what Dr. Herman calls "some contingent, but improbable dangers." None the less do I hold to a second opinion that the most quiescent fibroid requires watching. Our duty to the patient is to tell her so, and to induce her to remain under our own observation, or under the care of another who is in a position to watch and write down notes of the growth or diminution of the tumour. We must impress upon her mind the truth that the fibroid is doing no harm. We must further warn her that it may do a great deal of harm some day. But we must never frighten her by conjuring up the bugbear of cancerous degeneration or the certainty of a speedy increase of the tumour to colossal proportions. We know that the chances of the first complication are extremely rare, and the risk of the second but slight; therefore in wilfully magnifying such possibilities we should be guilty of gross prevarication.

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[1] "An Address on some Points in the Natural History of Uterine Fibroids." *Lancet*, Vol. I., 1900, p. 147. "The Past and Present Treatment of Uterine Fibroids." *Brit. Med. Journ.*, Vol. II., 1902, p. 1153.

Since the above paragraph was written Mr. Mayo Robson remarked in his Address in Surgery at the Swansea meeting of the British Medical Association: "As usual, the best course is the middle one; many cases need no treatment, whilst others, when growing rapidly or when associated with free bleeding or pressure-symptoms, demand operation." Professor Stephenson declared at the same meeting that he had watched cases where women had carried fibroid tumours for 30 and 40 years with little inconvenience. Professor Howard Kelly recently observed: "I note with regret to-day an increasing tendency to sacrifice small myomatous uteri, which in my own judgment either require no operation at all, or, at the most, call for myomectomy." ("The Selection of Methods in Abdominal Hysterectomy." *Journ. of Obstet. and Gyn. of the Brit. Empire*, Oct., 1903, p. 343.)

In considering any patient's condition and interests, we must be very wary about the opinions of others, however authoritative they may seem. The experienced physician and the successful operator can never be wholly free from bias, and in this case they ever tend to oppose each other. The physician has treated women in comfortable circumstances, counteracting to the best of his ability the effects of years of uterine hæmorrhage and the discomforts of a bulky tumour. His patients may feel reasonably pleased with him for his treatment; nevertheless many of them would undoubtedly have been the better for early hysterectomy. Yet a doctor in his position cannot be expected to recognise that truth. The keen operator is prejudiced in the opposite direction. He shows his numerous recoveries and his low mortality, and is sure that recovery is full proof that the operation was necessary and justifiable. Yet a woman who has recovered from hysterectomy for fibroid may become a victim to subsequent complications which will entail far more discomfort than that which was caused by the presence of the tumour.

The opposing arguments which the physician and surgeon can bring forward must be duly scrutinised. The surgeon can rightly urge that the patient who has submitted to operation feels free from the burden of mental worry caused by the presence of a tumour. This burden is no fiction. Several of my own patients have volunteered the declaration, long after convalescence, that they were restored to a peace of mind which they had lost for years. As long as the tumour is present, after the patient has become aware of its existence, she cannot feel quite easy. A successful operation, then, usually means the restoration of mental comfort to the patient, but it involves many more benefits. The tumour being removed, the patient is safe from numerous troublesome, pernicious, obstinate, and sometimes dangerous complications to which the growth might have given rise had it not been extirpated. Supposing that such complications had already existed, and consequently harassed the patient before hysterectomy was performed, the operator can feel fairly sure a year later that, had the operation been postponed to that date, its risks would have been greater. This assurance will apply far more surely to each succeeding year, even for some time after the menopause, for, although the newer theory may have been pressed too far, Kleinwächter, its earliest supporter, was quite correct when he insisted, on wide clinical experience, that the change of life does not necessarily cure fibroids.

On the other hand the physician who opposes operation when the tumour is unassociated with any complication, and is of small dimensions, has arguments of considerable force in his favour. The growth of a fibroid is not deadly, as is the case with a cancer; nor is it regular and certain to entail complications, one of which must at length prove fatal, as is the case with an ovarian tumour. The impossibility of calculating the future growth of a fibroid is put too much in the background by the advocates of hysterectomy. The operation itself is, at the best, no light experience, for even under the most skilful and kindly nursing the first few days are distressing to the patient. Hernia of the cicatrix, to be presently discussed more fully, is ever a possible after-result. Intestinal obstruction, the most perilous of all complications, is always possible, whatever method of operation be adopted. Another sequel of hysterectomy, sometimes occurring a year or more after convalescence, is the discharge of ligatures in very inconvenient directions. After enucleation of a fibroid from the posterior layer of the broad ligament many ligatures have to be applied to bleeding points in the pelvic connective tissue. Infection of these sutures by the Bacillus coli is always possible, for connective tissue, unlike peritoneum, is not very tolerant of foreign bodies.

The gravest possibility, however, when hysterectomy has been performed, is the advent of psychical disturbance. Hence close enquiry should be made about the patient's previous history; and even when that has been done, it may turn out that her friends have deceived us. The question of the management of the ovaries is, in respect to this complication, specially important; it will be discussed presently. Here it is sufficient to bear in mind that hysterectomy for fibroid is decidedly more dangerous than double ovariotomy when the patient's mental condition is unsatisfactory.

Such are the more important arguments for and against operation when a patient has a fibroid which is not threatening to give trouble. A few words may be said about treatment where an operation does not seem urgent.

This "treatment" is purely symptomatic. Light, nutritious diet, with as little red meat as possible, is well suited to such cases. Certain mineral waters are of indirect service. The advocates of electricity claim triumphs, but its application requires great experience, whilst the results are often doubtful, and in some cases which I have observed distinctly bad. When bleeding is present, ergot is not to be despised, especially if given in moderate doses with the ingredients so mixed that the active principle is not decomposed long before the physic is swallowed. There are fortunately means of administering ergot with something approaching precision. Large doses in any form are harmful, and when cardiac disease is present ergot is contra-indicated.[1] Chloride of calcium is vaunted by some authorities, but its method of action is uncertain, and the benefits derived from it doubtful. An abdominal belt, to support the tumour anteriorly, is always advisable.

Thus "treatment" is simple enough, for we may safely discard a very large number of remedies in which authorities (and others) have put their trust on insufficient grounds. As long as the patient keeps well, the tumour will keep well as a rule. But the exceptions are large; and the growth may take on certain well-known changes, such as sudden increase in size, involving severe pressure-symptoms, or it may give rise to exhausting hæmorrhages or to other harmful complications.

Let us, therefore, now consider operative treatment, which is demanded at once when bad complications are present, whilst its necessity is ever within the range of possibility when a fibroid is still without any other symptom besides its presence.

Not many years ago the operation of hysterectomy was attended by a frightful mortality. The practice was introduced of amputating the ovaries and tubes in cases of bleeding fibroids to check the hæmorrhage and arrest the growth of the tumour; and with the latter aim the same operation was undertaken

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[1] *See* "The Relations of Organic Affections of the Heart to Fibro-Myoma of the Uterus." By Dr. T. Wilson. *Trans. Obst. Soc. Lond.*, Vol. XLII., 1900, p. 176. This excellent treatise deserves the full attention of every practitioner.

in cases where there was no escape of blood. Its advocates insisted that no ovarian tissue must be left behind. In this theory lay the weakest point of the operation, when duly analysed as to its technique and to its after-results. I have endeavoured to make clear elsewhere[1] that, owing to the anatomical relations and the structure of the ovarian ligament, it is in most subjects impossible to avoid leaving some ovarian tissue behind. Yet I have had eminently successful results, when a small piece of ovarian tissue had unavoidably been left on both sides. It not rarely happens that neither ovary can be removed, owing to their relations to a big, vascular fibroid; often 10 or 15 years ago one was amputated and the other found to be irremovable. I have followed up several cases where one ovary only could be taken away, and in more than one the results were quite satisfactory, for bleeding ceased and the tumour diminished in size. The theory that in such cases the ovary left behind was physiologically dead, being flattened out under the fibroid and consequently atrophied, I know to be quite invalid, for in some of the cases in question the ovary was not flattened out, but projected amidst a mass of engorged veins from the surface of the tumour, whilst I have noted that ovaries squeezed out flat between a big tumour and the bony pelvis may be full of follicles, and contain normal corpora lutea. One limitation admitted by the advocates of oöphorectomy for fibroids was the situation of the tumour in the uterus; if in the lower segment or cervix, removal of the ovaries would, they maintained, be useless, since a new growth in those parts is supplied by the uterine artery, whilst it is the ovarian vessels that are tied in this operation. It happens that I have had good results in more than one case where the growth was in the lower segment and the fundus free. On the other hand I have seen the most careful amputation of the appendages in cases where the fibroid lay above the lower segment followed by no good result. The frequency of what may justly be called "cure" in cases where the principles of the operation were not, or could not be carried out, has convinced me that the benefits of oöphorectomy for fibroids were largely due to some other factor than removal of the ovaries. That factor has not yet been revealed. Certain eminent authorities[1] still practise this operation, claiming good results, but precise statistical evidence is wanting.

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[1] Harveian Lectures, Lecture I., *Lancet*, February 7, 1903, paragraphs headed "Surgical Anatomy of the Broad and Ovarian Ligaments" and "Oöphorectomy for Fibroids and its Teachings." These lectures include a summary of my own operative and clinical experiences in the treatment of fibroids.

When the tumour itself is to be removed, it may be enucleated through its mucosa and drawn out at the vagina as though it were a polypus. Should this procedure be impracticable or inadvisable, there remain several operations termed myomectomy, vaginal hysterectomy, supra-vaginal hysterectomy and panhysterectomy.

The first is logically the best, being essentially conservative. When the tumour is a pedunculated, sub-peritoneal growth, it may be treated like an ovarian tumour. It is not always easy, however, to stop hæmorrhage effectually, and it occasionally proves safer to fix the stump of the pedicle to the lower angle of the abdominal wound than to let it go back into the peritoneal cavity. When the tumour is sessile, myomectomy means enucleation. The main objection to enucleation is its difficulty and risk. An expert accustomed to operations on the female organs and with all the advantages of an aseptic technique at his disposal may succeed in enucleating fibroids of considerable size through long incisions made in the uterine wall, but the operation is not satisfactory in the hands of less experienced operators.

The propriety of removing a uterus, subject to a small fibroid, through the vagina is still under debate. Many teachers urge that, when the uterus and tumour form a mass small enough to be removable in that manner, no operation is justifiable. This objection, we must remember, does not apply to little fibroids which cause much bleeding or give rise to general debility through necrotic changes. This operation is not always practicable, so that the operator may find himself obliged to make an abdominal wound in order to remove the diseased uterus. It must be allowed that experts in vaginal surgery have had good results in the practice of this operation, but too much has to be performed in the dark.

Panhysterectomy is ideal as regards completeness. The cervix is removed and therefore cannot become the seat of cancer, nor can its stump slough, or undergo other pernicious changes owing to infection of ligatures in its substance, or be the cause of intestinal obstruction through adhesions. Its advocates, on doubtful grounds, consider that there is less danger from hæmorrhage than in supra-vaginal hysterectomy. But panhysterectomy is a long operation even in the hands of experts.[1] The after-results are not always satisfactory. I examined, a few years ago, a healthy young woman, aged 25, who three months previously had undergone panhysterectomy at the hands of an eminent foreign operator. Painful contraction of the vagina had developed and there was much pricking in the cicatrix. I have heard of similar complications in other cases. As a good deal of tissue in the pelvis is sewn up, leaving a ridge in the pelvic cavity, dangerous adhesions are always possible. It must be admitted, however, that there is less risk of this complication than when the cervix is not entirely removed. With more experience surgery may establish panhysterectomy as a valuable mode of procedure, but it is not yet in universal favour.

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[1] Murdoch Cameron, *loc. supra cit.* Gerald Garry claims to have observed very bad after-results. *Brit. Med. Journ.*, Vol. II., 1902, p. 1372.

The supra-vaginal operation with the serre-nœud or elastic ligature was once very popular and still has a few advocates. Unfortunately the wire was an instrument of torture as more than one patient informed me. The stump sometimes sloughed, so as to infect the peritoneum, and a bad hernia often developed in the lower angle of the abdominal wound.

Altogether, the retro-peritoneal variety of supra-vaginal hysterectomy is the simplest and best operation for the removal of a fibroid uterus. The term "retro-peritoneal," or "sub-peritoneal," is advisable, for the stump is placed behind or under the peritoneum, outside its cavity. I admit that this principle of making everything concerned with the stump as extra-peritoneal as possible must be qualified; thus, it is better that the blood which always oozes from the stump for a few hours after the operation should flow into the peritoneal cavity than accumulate in the pelvic connective tissue. Still, when the operation is thoroughly successful, the stump becomes an absolutely retro-peritoneal or sub-peritoneal structure.

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[1] Herbert Spencer: "Total Abdominal Hysterectomy (especially by Doyen's method) for Fibromyoma Uteri; with Notes of 14 Cases." *Brit. Med. Journ.*, Vol. II., 1902, p. 1131.

A detailed account of this operation would be out of place here as this article deals with our knowledge of the treatment of fibroids, whilst retro-peritoneal hysterectomy is spoken of here mainly as the best surgical procedure, taking all things into consideration, when radical measures are demanded. A few words, however, may be said about certain factors of importance in regard to the operation, namely, the abdominal wound, the uterine flaps, the treatment of the ovaries, and the question of sutures.

The abdominal incision would seem to demand no special notice, seeing that it is made in other operations besides hysterectomy. We may profitably bear in mind, however, the truth that the parietes are usually very vascular in fibroid uterine disease; hence bleeding vessels should be carefully secured by pressure-forceps left on for a few minutes. Silk ligatures are, for evident reasons, objectionable. The incision should always be made long enough to allow of the extraction of the fibroid without bruising of the recti.

The uterine flaps need not be cut long; it seems best to fashion them of a length just sufficient to include a little of the tissue of the body of the uterus—mucous membrane as well as muscular wall. This method is in accordance with a theory on which more will be said in relation to the treatment of the ovaries in hysterectomy. We must remember that the Americans, and many British operators, favour amputation below the level of the os internum, or, in other words, through the cervix. No endometrium is left behind, as it does not extend below the os internum. Personally, I find that the patients, as a rule, do very well after cervical amputation.

As these notes are devoted to the present position of our knowledge with regard to the treatment of uterine fibroids, we must be careful to define the present position of our ignorance about the same subject. The "flap" question now under consideration and the right course with respect to the ovaries remain unsolved problems. I have designedly implied above that, though it would seem best to save some of the endometrium or mucous membrane of the body of the uterus, experience shows that cases fare well when none is left, Theories on this or on any other disputed point of treatment based on the low mortality of some fifty or a hundred hysterectomies by one clever operator are seldom, if ever, convincing to others. The successful surgeon is often strongly biassed as to the value of some detail in treatment which he adopts, and hence may ascribe good or bad after-results to a wrong cause. We read of one operator attributing his good results in a series of abdominal sections to his practice of flushing the peritoneal cavity with saline solution, whilst another argues that his equally-satisfactory results are due to his principle of drying the peritoneum thoroughly in every case. The same fallacy underlies arguments about flaps. It is certainly a mistake to cut very large flaps of uterine tissue, although they often give no trouble. But the share which the practice holds in respect to good and bad after-results can hardly be estimated with precision. In one case the uterine tissue may be healthy, in a second unhealthy, and in a third infested with small interstitial fibroids, the necessary enucleation of which involves much bruising of the flaps.

I have expressed an opinion above that the treatment of the ovaries in hysterectomy is a problem not satisfactorily solved. The older authorities who still operate maintain that it matters little whether the ovaries be left or removed. The younger school consider that it is a duty to save them. There remain others, myself among the number, who consider that experience shows that, taken as a whole, it is best to leave one ovary at least, provided that it be perfectly healthy, but that the advantages of the conservative treatment of the ovaries have been exaggerated, and that the ovaries have been known to give trouble after convalescence appeared complete.

Abel and Zweifel insist that not only must the ovaries be left, if we desire to save the patient from speedy and acute symptoms of the menopause, but that a portion of the endometrium or mucous membrance of the uterus proper must be saved as well. This theory is of course an argument in favour of long flaps In my own practice I find that the saving of a portion of the endometrium, that is to say, amputation above the level of the os internum, distinctly, though not very markedly, influences, in a favourable sense, the catamenial functions. For that reason I observed above that it seems best to fashion the uterine flaps of a length just sufficient to include a little of the uterus above the cervix.

Reference has already been made to psychical disturbance after hysterectomy. It is by no means unknown, and its possible occurrence favours the conservative treatment of the ovaries. The precise danger, however, of removing both ovaries can never be estimated from statistics. Post-operative insanity has been known to follow many surgical procedures besides hysterectomy, whilst in several cases of that operation, in which it occurred, the patient had been subject to attacks of insanity already, although the operator had not been informed of the fact. Neurotic symptoms occurring during the menstrual period would seem to indicate that removal of the ovaries would make the patient more neurotic. Yet in two cases in my own practice where I removed both ovaries with large fibroids, the patients had been subject to syncope at the period quite independent of hæmorrhages; nevertheless the fainting-attacks disappeared entirely after the operation.

We must also remember, in our anxiety to avoid a stormy menopause, that some women, when it occurs prematurely owing to removal of the ovaries, care little about their flushings and faintings, just as many subjects trouble little about these symptoms when they set in naturally and at the usual age. Others worry under an artificial, just as under a normal, menopause about the slightest flushings even at long intervals.

Thus evidence seems rather in favour of the conservative treatment of the ovaries; yet its advantages at the best are certainly not great, and in no individual case can they be accurately estimated. Unfortunately evidence has also proved that there are disadvantages associated with this treatment. A piece of Fallopian tube left with an ovary may inflame or even suppurate. The spared ovary itself may become the seat of a new growth. It is certain that an inflamed ovary and the tube should always be removed; and when the inflammatory disease affects both appendages, the operator should never scruple to remove them.

Enough has been said to show that the present condition of our knowledge about the effects of removing both ovaries is by no means satisfactory. Several hundred after-histories are required, and they should be collected by practitioners and registrars who are in a position to watch them for many years. A single series reported by one operator and his assistants is of small value for many reasons, and can never convince those who differ from the reporter as to his theories and practice. Credit, nevertheless, must be awarded to all who have published such reports[1] as, however unwittingly prejudiced these operators may be, they have added much to our knowledge of the physiology of the ovaries.

The abdominal wound has to be closed, and we ought to adopt that method which will ensure a firm cicatrix so that no hernial pouch can form. These herniæ give rise to great discomfort, which we must take into account, since many fibroids suitable for different reasons for operation do not make the patient feel uncomfortable. The herniæ are also areas of possible danger, firstly through incarceration of intestine—strangulation is very improbable—and secondly through sloughing of the thinned sac, a complication usually fatal; but in women in easy circumstances these ventral herniæ rarely cause more than discomfort.

Unfortunately, although we ought to adopt the method of suture which will absolutely ensure a firm cicatrix, that method remains unknown. Deep sutures of silkworm-gut or silk including all the layers of the wound often ensure perfect union, provided that they are not tied too tightly or placed too close together or too far apart. Care must also be taken not to include more than a quarter of an inch of the peritoneum on each side, especially in a thin subject. Notwithstanding all precautions, however, a bad hernia may develop.

Suture in layers, the opposite method, is popular at present, but it is open to great objections. It involves the leaving of a foreign body in tissues disturbed by respiration and other movements. Early or late suppuration is not unknown. The records of registrars and of medical societies bear witness to this truth;[1] and I know of several such cases which have occurred within the last twelve months and have not as yet been reported; and I must add two which I have observed in my own operative practice. The supporters of this method of suture may remind us, I must admit, that suppuration is not necessarily followed by hernia. Ten years ago I removed a double pyosalpinx from a very fat, unhealthy patient, using Keith's drainage-tube. The wound suppurated, yet the cicatrix is at the present day perfectly firm and there is not the least trace of hernia.

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[1] Especially to Dr. Crewdson Thomas. *See* "The After-Histories of 100 Cases of Supra-vaginal Hysterectomy for Fibroids," *Lancet*, Vol. I., 1902, p. 294. A series of 1,000 prepared with the same care would be of great value. Statistics of 44 cases in my own practice will be found in the second Harveian Lecture, *Lancet*, February 14, 1903.

Altogether, carefully-applied deep interrupted sutures do best. In very fat subjects, where it is not easy to bring the cut edges of the sheaths of the recti into apposition, a few interrupted fine silk sutures are advisable. Catgut is apt to yield too soon, except when chromicised, and then it may act like a foreign body.

I have dispensed altogether with statistical tables which have been freely made public during the past ten years. They deserve study, but are apt to be very misleading. Tables prepared by the operators themselves are untrustworthy in demonstrating whether hysterectomy for fibroid be justifiable, just as they afford no certain proof of the superiority of one particular method, as has been already noted. The present position of our knowledge about hysterectomy is that, if it be performed on a hundred cases where the fibroid is small or of moderate size and gives rise to no severe symptoms, it is conceivable that all may recover, especially should the operator happen to be an expert who had removed many abdominal tumours before he undertook the first of his hundred hysterectomies. These good results in simple cases are used as an argument in favour of operation, whilst on the other hand the opponents of hysterectomy as routine treatment look upon a very low mortality rather as a reproach than a credit to the operator, implying that many hysterectomies in a brilliant series were quite unnecessary.

We must not forget that statistics of abdominal operations originated when Spencer Wells undertook those labours which played such a large share in the establishment of ovariotomy as a justifiable surgical procedure. The removal of an ovarian tumour was shown to be necessary, to be practicable, and to be fairly safe should certain precautions be taken. When Wells began his first series, Baker Brown had given up the operation ; very few others were attempting it, and most men were lapsing into the old state of indifference, if they were not loudly protesting against it.[1] Current statistics about hysterectomy for fibroid are based on very different conditions both in respect to the nature of the disease and the attitude of the profession towards abdominal operations. Nowadays nobody can deny with reason that an ovarian tumour should be removed, save under certain quite exceptional circumstances ; the same cannot be said of uterine fibroids. At the same time hysterectomy for fibroids is largely practised. It was the opponents of ovariotomy—an operation then rarely attempted—who were in a majority when Wells published his earliest statistics. At the present time when tables of hysterectomies are so largely published, it is the detractors of that operation who are in a minority. The old statistics of Wells convinced and converted the opponents of ovariotomy. The new statistics of hysterectomy fail to convince its opponents, who do not deny the low mortality, but who ask why the operators wage war against all fibroids small or great."[2]

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[1] *St. Bartholomew's Hospital Reports*, Vol. XXXV. (1899), p. 26; *Middlesex Hospital Reports* for 1897, Case XXIV., pp. 222, 223 (suppuration within three weeks); *Trans. Obst. Soc. Lond.*, Vol. XLIV., 1902, p. 183 (a year after operation). In all these cases the operators were surgeons of great ability.

Statistics of hysterectomy, however, must be fairly judged on their own merits. They should never induce us to operate upon every fibroid that henceforth comes under our care. They may reasonably fail to convince us that every case which they include was the better for operation. Still they show that recovery is almost certain when a fibroid of a moderate size is removed by an experienced operator. That the patient is always in certain respects a gainer by the removal of her fibroid, I have already demonstrated. In the worst cases, where operative interference is most necessary, the mortality remains considerable. These facts must be seriously taken into account by all who have cases of fibroid disease of the uterus under their charge.

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[1] Spencer Wells: *On Ovarian and Uterine Tumours: their Diagnosis and Treatment*, 1882, p. 196.

[2] Murdoch Cameron, *loc. jam cit.*

COMPLICATIONS MET WITH DURING AND AFTER OVARIOTOMY.

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(With Plate XVIII.)

ALTHOUGH the number of cases which form the basis of this paper make up only a small series, nevertheless they possess so many points of special interest that the writer has thought that it might be of interest to put them on record under the above title.

A.—The following are among the complications met with at the time of operation:—

(1) Pregnancy; (1A) Vesicular Mole; (2) Carcinoma Recti; (3) Twisted Pedicle; (4) Rupture, and results of repeated tapping; (5) Adhesions; (6) Burrowing into the Broad Ligament and into the Meso-sigmoid.

B.—Of the conditions which may disturb convalescence, those mentioned below are more especially referred to:—

(1) Vomiting; (2) Sinus-formation; (3) Parotitis; (4) Ileus paralyticus; (5) Embolism (pulmonary); (6) Thrombosis (pelvic); (7) Bronchitis; (8) Pneumonia.

A.—COMPLICATIONS DURING OVARIOTOMY.

(1) *Pregnancy.*—Since ovarian disease is commoner during the fertile period of a woman's existence than either before or after, it is only natural to anticipate that ovarian tumours will sometimes be found to complicate pregnancy, labour, and the puerperium. That ovarian tumours and pregnancy are not more often associated may be due to the fact that ovarian disease is an undoubted cause of sterility. Pregnancy is one of the rarer complications with which one meets in the performance of the operation of ovariotomy. From the investigations of Flaischen, Lohlein, and Tarnier, McKerron estimates that one pregnancy in every 2,500 is associated with ovarian tumour. The conclusions arrived at by recent writers are that pregnancy appears to prevent, rather than to cause, disease of the ovary;

and it neither accelerates the growth of ovarian tumours already present, nor does it favour malignant changes in simple or multiple cysts.

The concurrence of pregnancy and ovarian tumour must be looked upon as a grave condition, as regards the prognosis both for the mother and for the child. The mother's life is in danger from such complications as rupture of the cyst, suppuration, and twisted pedicle. The life of the offspring is imperilled by delayed or obstructed labour in cases which have gone to term, and where pregnancy or labour is found complicated with a densely-adherent ovarian cyst. At the present time small tumours are regarded as being quite as great a source of danger as larger ones; and McKerron has shown that such complications as those mentioned above may be looked for in one out of every three cases.

The point to be desired above all others is an early diagnosis. If the tumour is removed, as it was in the case recorded below, before complications arise, the prognosis for the mother is scarcely more grave than it would be apart from the existence of pregnancy; and, as regards the child, I am convinced that the risk of abortion has been greatly exaggerated.

The tumour described below belongs to the commonest type met with as a complication of pregnancy, *i.e.*, a multilocular *cyst.* Solid tumours are a very rare association of pregnancy, and dermoids also occur less frequently than simple or multilocular ovarian cysts.

As regards position, the smaller ovarian cysts usually lie against the sacrum in the pouch of Douglas (as in my case); here they may become incarcerated, as was the case with a dermoid recorded by Dr. Routh (*Obst. Trans.*, Vol. XL.). In such instances the danger from obstruction to the passage of the child is obvious. Another danger from the same cause—a small tumour in the pelvis—must not be overlooked, viz., incarceration of the uterus, and abortion. Abortion may be brought about in yet another way; if the uterus escapes impaction, it may undergo torsion, and this, by impairing the circulation, may lead to expulsion of the uterine contents. Again, if pregnancy goes to term, deviation of the uterine axis may prevent the head from engaging in the brim. Even sacculation of the uterus seems to have resulted from the presence of an ovarian cyst.

In a case of vesicular disease of the chorion in Mr. Malcolm's wards at the Samaritan Free Hospital, a fortnight after removal of the mole, abdominal section was required for acute symptoms due to the twisting of the pedicle of an ovarian tumour; both ovaries were found to be the seats of multilocular cystomata. The not-infrequent association of ovarian disease with hydatidiform mole has frequently been recorded, especially in German literature. I have met with nine other cases in the literature on this subject, seven German, one recorded by McKerron, of Aberdeen, and another by Wilson (*Lancet*, 1840). Although Findley will not allow that cystic disease of the ovary has any causal relation with cystic degeneration of the ovum, Marchand says that "the influence of the maternal organism makes itself felt upon the ovum in the ovary, the latter transmitting any pathological peculiarities it may possess to the developing ovum." For a fuller discussion on this interesting subject the reader is referred to a paper written by Mr. Malcolm, Dr. Bell, and myself, upon the above case, and published in the *Trans. Obst. Soc.*, Lond., 1903. For the present purpose enough has been said to show that the influence of ovarian disease on pregnancy is of serious import, and to prove that the complication calls for immediate treatment.

With reference to symptoms, they may be absent entirely, but the most constant is pain, which is generally referred to the ovary from which the tumour springs, although (*vide infra*) it may be chiefly on the opposite side. This pain may be of a dragging character and continuous, or be sharp and sudden.

When the tumour lies on the sacrum, constipation is complained of, as in the case recorded below. Irritability of the bladder is not an uncommon symptom. When the tumour is in the abdomen, the unusual size, not in keeping with the period of gestation, may first arouse suspicion. Sudden severe pain has resulted from a pelvic growth suddenly being displaced into the abdomen, with the tearing of adhesions, due to a jolt. When once it has gained the abdomen, respiratory embarrassment and general signs of pressure may follow.

The diagnosis of ovarian tumour complicating pregnancy may be very easy, or extremely difficult. Want of space will not allow me to do full justice to differential points; but a most systematic investigation of all patients with tumours of the ovary, if seen during their child-bearing period, should be made, in view of the possibility of pregnancy; and the same may be said of all cases of pregnancy.

The method of drawing down the cervix with a volsellum, whilst proceeding with abdominal palpation, is worth remembering, and much help may be got by rectal examination, which enables the examining finger to reach higher up the pelvis. In great abdominal distension tapping has been advocated as a help to diagnosis, but this endangers the uterus, and I should not advise its employment. Conditions which may be mistaken for ovarian tumour and pregnancy are:—Fibromyoma uteri, retroverted gravid uterus, floating kidney, combined extra- and intra-uterine gestation, hydramnios, and pelvis exostosis.

As stated above, the prognosis is mainly influenced by early diagnosis and the subsequent mode of treatment.

The treatment of ovarian tumour complicating pregnancy up to the year 1880 resolved itself into non-interference in the absence of urgent symptoms, tapping the cyst, and the induction of premature labour. In spite of the fact that experience has accumulated slowly since that date, records are now sufficiently numerous to prove by comparison that the soundest treatment is *immediate ovariotomy*—indeed, this operation is even more urgently demanded than it is when pregnancy is absent. The route by which the operation is carried out will vary with the size and position of the tumour. Wherever practicable, as in cases where the cyst is non-adherent and lying in the pelvis, I regard the vaginal route as the more suitable, since it obviates the risk of ventral hernia consequent upon the stretching of the abdominal scar by the steadily-increasing size of the abdomen during the remaining period of pregnancy.

The following are details of a case of ovariotomy performed through the vagina at the tenth week of gestation:—

M. T., aged 23, came to Mr. Handley's out-patient department at the Samaritan Hospital on February 12, 1903, complaining of pain in the left ovarian region "going through to the back," and becoming worse on walking. She had had one normal labour in July, 1902. Menstruation began at 13 years of age; it usually lasted 5 days, and occurred every 30 days; two diapers were used daily. The last period occurred on January 25, 1903. Micturition was not disturbed, but constipation had been a source of trouble. At the first visit Mr. Handley found the patient suffering from colitis, the result of using purgative drugs. Upon examination the uterus was normal in size and position. In the right posterior fornix was a fluctuating lump, palpable bimanually, the size of a man's fist. The surface of the lump was irregular; it was "somewhat mobile"; there was no inflammatory thickening. The diagnosis made was "small right ovarian cyst; colitis from purgatives." On February 19 the patient was much better, there was no pain; the lump was fixed and could not be pushed up even in the knee-elbow position. It did not press hard against the sacrum. On May 18, when examining the patient in the ward, I found the uterus the size of a two months' gestation, normal in position and mobile. In Douglas's pouch a very tense, irregular, fixed, cystic swelling was felt; it mainly presented in the mid-line, but reached to the right side. It was tender on palpation, which made me think that there was plastic peritonitis present. The temperature on admission was 99° F. I regarded the tumour as ovarian in origin and the uterus as gravid.

On May 22, with Mr. Handley's assistance, I opened the pouch of Douglas and removed a multilocular ovarian cyst and part of the right Fallopian tube. The cyst was readily withdrawn after puncture with Martin's trochar and dilator. The pedicle was easily ligatured, and a separate ligature was passed around the ovarian artery which showed prominently in the stump. The posterior vaginal wall bled very freely as the result of the associated pregnancy, but the hæmorrhage was stopped by oversewing the edge of the vaginal incision and the peritoneum of Douglas's pouch all along the whole length of the wound by a running catgut stitch. I was undecided whether or not to close the opening made in the posterior *cul de sac*, but finally decided to drain. For this purpose I lightly packed with 10-per-cent. iodoform gauze, which was kept in for three days. The cyst was of the size of a cocoanut, and contained three separate loculi; the fluid was of a pale straw-colour. The ovarian fimbria of the right tube was attached to the outer and upper aspect of the cyst. The convalescence was apyrexial and uneventful, the wound rapidly healed, and at the present time the pregnancy is running a normal course, there being still six weeks more to elapse before full term. The cyst is shown in Fig. 1.

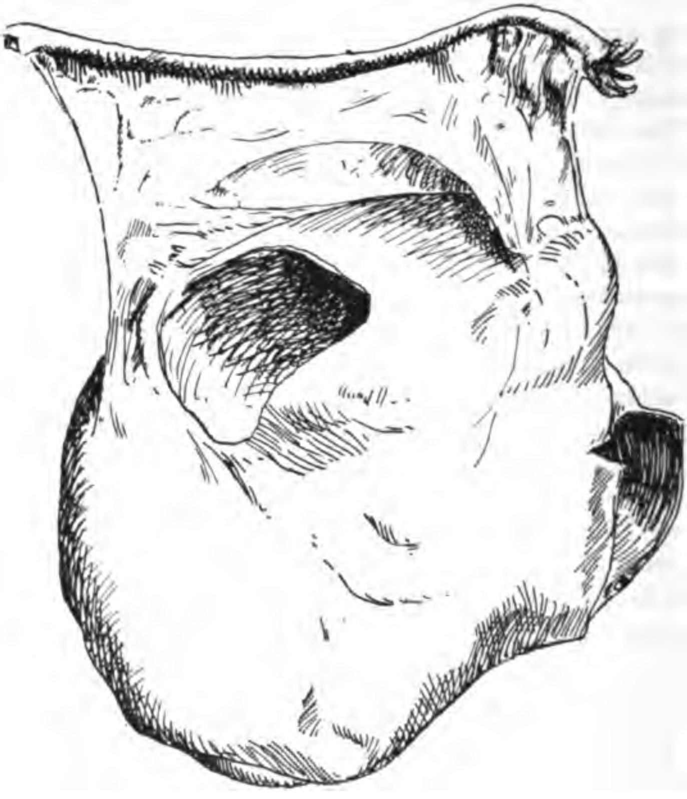

Fig. 1. *Ovarian cyst removed at the tenth week of gestation.*

(2) *Carcinoma Recti.*—My experience of this complication is interesting and instructive, inasmuch as the growth could not be discovered before removal of the semi-solid ovarian tumour, although the symptoms pointed to a rectal carcinoma, and the bowel was explored by digital examination. As the notes of the operation show, the rectum was displaced by a hard, solid fibro-adenomatous mass, arising from the hilum of the ovary, and this fully accounts for the growth, which existed in its second portion, being pushed out of the reach of the

examining finger. Carcinoma of the bowel is not an uncommon association of ovarian tumour. During the last eighteen months there have been two examples of this condition in the Charing Cross Hospital under the care of Mr. Stanley Boyd. In each case the main symptom was constipation, and in one there was a history of obstruction prior to admission. In each, the ovarian growth was benign, and in both cases the malignant tumour was situated in the splenic flexure. In neither instance was the malignant growth suspected beforehand or discovered at the time of removal of the ovarian tumours. The bowel-symptoms were attributed to pressure of the tumours. In one case acute intestinal obstruction supervened 48 hours after the ovariotomy, and an exploratory laparatomy revealed the cause. In the second case chronic intestinal obstruction continued after the ovariotomy; the diagnosis became obvious, but the exact site of the growth was not manifest until the autopsy some four weeks later. For good reasons a further operation was not performed in this case, and there was no necessity for a colotomy.

Dr. Herbert Spencer, in the course of conversation, mentioned a similar experience in his own practice.

In my own case I failed even at the operation to discover the rectal growth, the reason being that the sigmoid and upper part of the rectum were loaded with hard scybalous masses, and although I palpated the entire distended bowel I considered the whole mass to be fæcal, and overlooked the tumour. It was not until after the operation, and when I found that the rectal symptoms were not relieved by removal of the large ovarian growth, that I was able to discover the cancer of the bowel by rectal examination. Colotomy was then performed (by Mr. Wareham), from which the patient made a good recovery, but at the present time she is gradually losing ground, and her end is not far distant.

The lesson to be learnt from such cases is obvious. No matter what the character of the ovarian growth may be, if constipation, rectal discharge, and hæmorrhage, or actual intestinal obstruction, obtrude themselves as symptoms, we ought to be on our guard for such a complication as malignant disease of the bowel, and not content ourselves by explaining away serious, acute, or chronic bowel-symptoms as due to mere mechanical pressure of the ovarian growth.

Case 2.—Benign ovarian tumour complicated by carcinoma of the second portion of the rectum.

J. F., aged 44, had been married twelve years. She had had one child nine and a half years ago. Two early abortions had occurred, the last being in December, 1901. The patient came to Plaistow Hospital on December 4, 1902. She had been under treatment for hæmorrhoids for one year, and had obtained relief from aperient pills and simple enemata. During the past year she had been in Dagenham Isolation Hospital for small-pox. When first seen by me at Plaistow she complained of "pain in the back passage with a discharge of blood and pieces of white stuff." Defæcation was difficult and never complete; much straining was necessary, the motion being at times tape-like in shape. The patient had to go to stool as often as ten times in a morning; sometimes blood alone passed, at others fæces in small fragments. Menstruation was regular, lasting three or four days; it was painful, and occasional clots were passed. Lately the flow had been pale in colour. The pain started in both iliac fossæ and ran down both thighs and through to the back. There had been some leucorrhœa, but never a blood-stained discharge between the periods. Examination revealed a growth in the pelvis of stony hardness and nodular on the surface, which was pressing upon and displacing the rectum. This hard growth was continuous with a spherical tense mass of cystic consistence which filled the pelvis below, and reached to the umbilicus above. The cervix uteri was pushed to the right; the os was patulous. The sound was not passed. I took the tumour to be a fibroid of the uterus which had undergone cystic degeneration in front and above, whilst its still-solid portion was pressing upon the rectum behind and setting up signs of rectal ulceration and obstruction. On December 12, 1902, I opened the abdomen by a 4-inch incision in the mid-line. The abdominal part of the cyst at once presented and burst over the peritoneal content on passing the hand into the abdomen; thin greenish fluid escaped. The pelvis was filled by a second cyst which was adherent to the floor of Douglas's pouch. Uniting these two cysts upon their posterior aspect was an elongated solid mass made up of three tuberous lobes with a crenated outline, which on section showed a smooth homogeneous surface like that of a potato. This mass lay posteriorly, and pushed the rectum inwards; it was what I had mistaken for a fibroid, and to its relation with the rectum I had attributed all the symptoms which were really due to the carcinoma of the bowel.

This growth, partly cystic and partly solid, belonged to the left ovary, and had stretched the two folds of the left broad ligament over its lower half. Its removal was effected by ligaturing the adnexa close to the uterus and tying off the expanded broad ligament in front of the tumour.

The sigmoid and rectum were distended by hard fæcal masses. The right ovary contained two cysts united by a fragment of atrophic ovarian tissue. There was a small pedunculated cyst in the right meso-salpinx. These, together with part of the right tube, were removed. The uterus, thus deprived of its supports, was fixed to the anterior abdominal wall, and after tying off some oozing adhesions on the floor of Douglas's pouch and flushing the abdomino-pelvic cavity with sterile Koch's solution, the abdominal wall was brought together by means of three layers of silk sutures. The patient recovered without a rise of temperature, but rectal obstruction, which would only partially yield to enemata, led to a further examination of the bowel, and then only, after removal of the ovarian growth, was the carcinoma recti discovered. Colotomy for chronic intestinal obstruction was performed with the result cited above.

The solid part of this ovarian growth presents points of especial interest. As already stated, the tumour is a fibro-adenoma, which in microscopical characters resembles a growth reported on in the *Obstetrical Society's Transactions* by Dr. Macnaughton-Jones, which was submitted to a special pathological committee. The description of that growth so aptly describes my own, that I quote it nearly word for word. "The tumour (part involved in the growth) consists chiefly of well-developed fibrous tissue, arranged in intersecting bundles; within this fibrous tissue are numerous widely distributed, well-defined spaces filled with epithelial cells. These spaces are irregularly oval or elongated, occasionally branching. . . . There is no sign of invasion of the surrounding fibrous tissue by

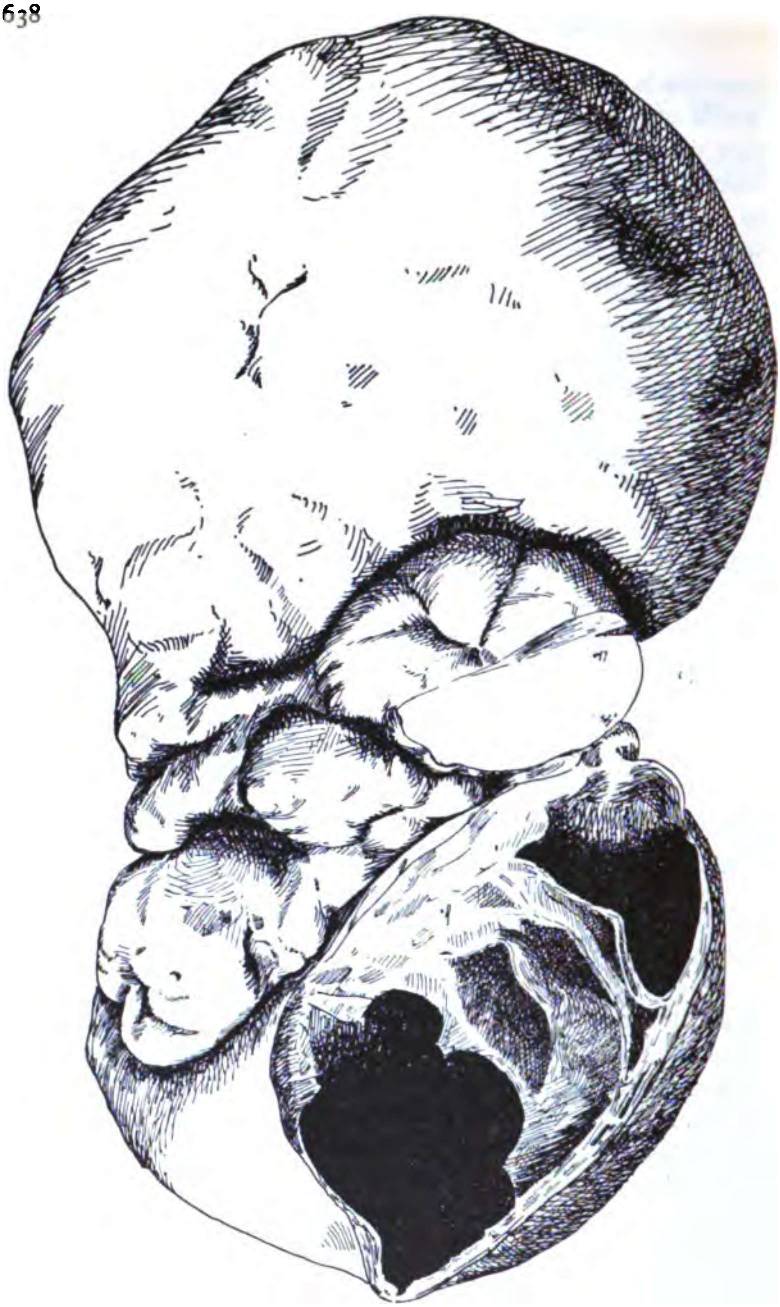

Fig. 2. *Multilocular ovarian cyst with solid dense fibro-adenomatous growth complicated by carcinoma recti. (a) Smooth cut surface from which microscopical sections were taken.*

epithelial cells and no small-celled infiltration. . . . The tumour is non-malignant, and in the arrangement of the epithelium it most nearly resembles that met with in some forms of adeno-fibroma of the breast." The only differences between my specimen and the one described above is that in the latter none of the alveolar spaces showed any lumina, whilst in mine the epithelium is columnar and so arranged as to show a central lumen giving the appearance of ducts ot various sizes cut across.

The microscopical characters of this tumour are shown in Fig. *2a*.

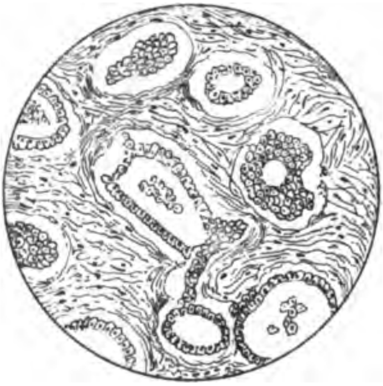

Fig. *2a*.

(3) *Twisted Pedicle.*—The next complication to which I desire to draw attention is that of torsion of the pedicle of an ovarian cystoma.

The cause of axial rotation of a tumour, with consequent twisting of its pedicle, has no adequate explanation. The movements of the bladder, the passage of fæces per rectum, and unusual exertion on the part of the patient have been suggested as causes. One thing is certain, that the complication is met with far more frequently when pregnancy is present than when it is absent; again, it is more prone to occur when two cysts are present. In a case, therefore, in which a cyst has undergone torsion of its pedicle, the condition of the uterus and that of the opposite ovary should not be overlooked, for a comparatively small ovarian cyst or an early pregnancy may be discovered, which had previously escaped observation.

Cases of torsion vary much in clinical signs, inasmuch as the degree of tightness of the twist, and the thickness of the pedicle, will determine whether the case is acute or chronic. In the latter condition the change may be so gradual that at no period in the patient's history had she ever experienced anything like a crisis, yet not infrequently the cyst is found by the operator to have been completely twisted off from its original attachment and lying as a necrosed mass, or still living by virtue of a blood-supply brought to it from other sources through the medium of adhesions.

Where, however, the pedicle is long and thin and the degree of torsion great, the veins rapidly become occluded, whilst for a time the arteries continue to pump blood into the cyst-wall. Extreme venous engorgement follows: from the wall of the cyst extravasation of blood takes place into its interior, and severe anæmia and even death may result from this alone. Later on, parts of the cyst-wall may necrose and rupture with hæmorrhage into the abdominal cavity, causing death.

The clinical signs of acute torsion are so severe as to be equalled only by a rupture of a gravid tube, strangulation or perforation of the bowel, and similar acute conditions, whilst a sudden enlargement of an abdomino-pelvic tumour, accompanied by intense pain and sickness, feeble pulse and cold extremities, would make the diagnosis evident. The treatment of such a condition can be summed up in two words—*immediate ovariotomy.*

Case 3.—Unilocular non-adherent ovarian cyst which had undergone axial rotation.

A married woman, aged 54, was admitted into St. Mary's Hospital, Plaistow, suffering from intense abdominal pain and extreme sickness, on August 23, 1901. She had been seen by Dr. Griffith at St. Bartholomew's Hospital a few weeks before admission, and she had refused his advice to undergo an operation for an abdominal tumour. The woman's condition seemed so critical to the medical officer that he telegraphed asking me to operate at once. On arrival I found the patient extremely ill; she had been vomiting freely for three days before her admission. Her temperature was 97° F., and her pulse 100; her extremities were cold. The abdomen was greatly distended and tender. I made no external examination as everything had been prepared for the operation and the abdomen was bandaged, but the cystic collection of fluid was recognisable in spite of the dressings. The woman at first refused operation, but her husband was sent for, and he prevailed upon her to submit. The operation presented no difficulties. The cyst had thick walls; it was of a deep, livid, maroon colour, and contained nine pints of dark, porter-like fluid. It was unilocular, and had a well-defined pedicle; this had two complete twists in a direction contrary to that of the hands of a clock. There were no adhesions and no patches of necrosis. The ovary was adherent to the cyst-wall; it was intensely engorged. The appendages were likewise very much congested, showing enormous vessels. The latter were nearly the size of one's little finger; the drawing (Fig. 3) is so reduced as to give no idea of their size. The cyst arose from the hilum of the right ovary. The patient's condition improved from the time of the operation; the temperature rose to normal, the pulse-frequency fell, sickness and pain subsided. Although the patient was very obese, she showed no sign of hypostatic congestion, and some respiratory frequency noticed before operation disappeared afterwards. A stitch-abscess was the only flaw in the convalescence.

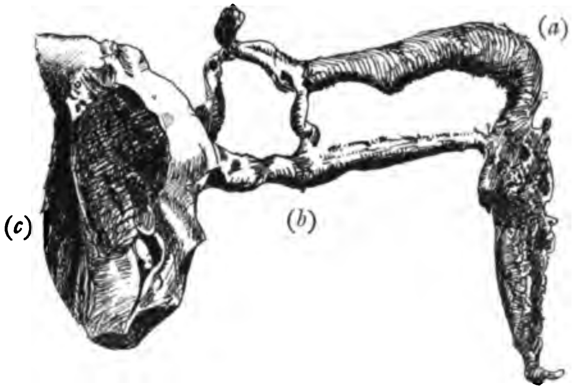

Fig. 3. *Appendages removed from Case 3. (a) Fallopian tube; (b) ovarian artery; (c) ovary engorged with blood. ¼ natural size.*

The cause of torsion in this case was never determined.

The woman had passed the menopause four years previously. She had been conscious of the presence of an abdominal tumour for two years. There was nothing amiss with the opposite ovary (left). This case bears out the well-known fact that the prognosis of ovariotomy for non-adherent tumours with torsion of their pedicles is good. The large cyst is figured in the miniature drawing (Fig. 3*a*).

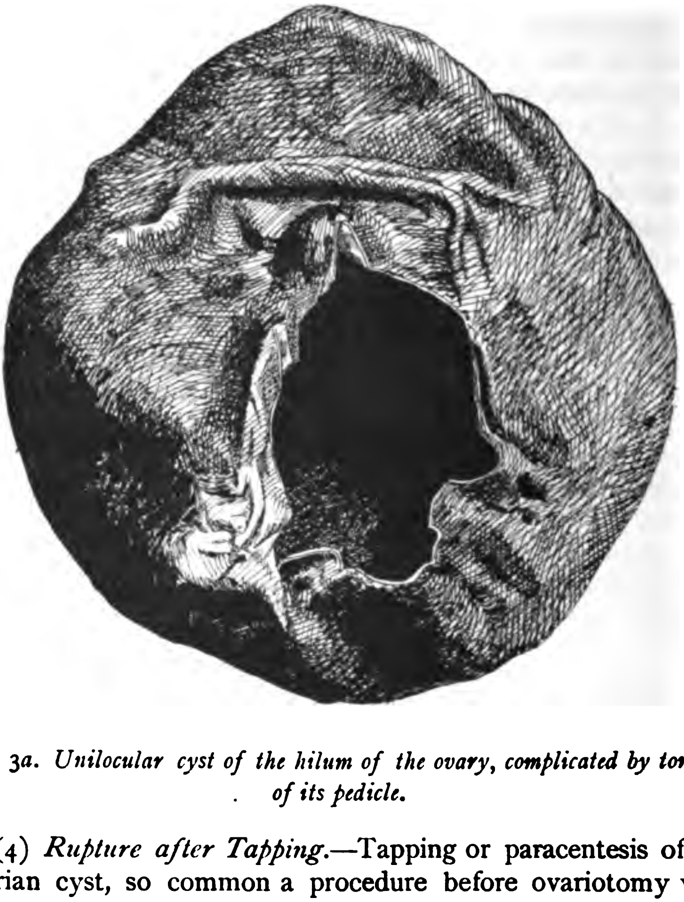

Fig. 3*a*. *Unilocular cyst of the hilum of the ovary, complicated by torsion of its pedicle.*

(4) *Rupture after Tapping.*—Tapping or paracentesis of an ovarian cyst, so common a procedure before ovariotomy was introduced, is now but rarely resorted to, even as a palliative measure. Whilst it does, without doubt, relieve distressing symptoms, such as dyspnœa and general respiratory embarrassment, it is not free from serious risks even when carried out with all antiseptic precautions. In the first place, if the cyst is

suitable for tapping, *i.e.*, if the fluid would run easily through a cannula, the tapping may result in its escape into the peritoneal cavity with a risk of peritonitis. This risk is even greater when the contents are colloid; and, moreover, it is well known that dissemination of papillomata has occurred in this way. Tapping predisposes to suppuration, to rupture, and to torsion of the pedicle (McKerron). When a patient is suffering from some intercurrent trouble, such as bronchitis or lymphatic and venous obstruction in the lower limbs, some writers still advise paracentesis. In the case recorded below, the patient was twice tapped by her doctor whilst in a cottage hospital, because, although she was suffering from bronchitis and swollen legs, she stoutly refused operation. The medical practitioner drew off 4½ gallons of cystic fluid before the patient was admitted into St. Mary's Hospital, Plaistow. There is little doubt that the tappings were largely responsible for the rupture of the cyst, and certainly for many of the extreme difficulties in performing the subsequent ovariotomy.

Case 4.—Ruptured ovarian cyst complicated by dense adhesions and a large para-umbilical hernia.

E. B., aged 63, had married 29 years ago, and had been a widow for 17 years. She had had twelve confinements, all easy, the first three at the seventh month. The first child was still-born; nine children were born at full term, and six were now living. The menopause occurred at the age of 48; she had lost no blood since. There was no vaginal discharge. Micturition was frequent, and the urine contained a trace of albumen. The bowels were regular. For the last 3½ years there had been increasing swelling abdomen. (A para-umbilical hernia had been present for 17 years.) The swelling had increased rapidly in size during the last 12 months. *There had been no pain.*

In April, 1903, the medical practitioner drew off about one pint of fluid. About a week later, when the patient was in a cottage hospital suffering from bronchitis, he drew off 4½ gallons of cystic fluid: this relieved the dyspnœa; it produced no difference in the quantity and character of the urine.

I did not see this patient until she was placed on the operation-table, but I made a rough sketch of her abdominal condition as then seen (*see* Fig. 4). The maximum girth was

48 inches. The abdominal walls overhung the lower ribs, and the upper 3 inches of the thighs, and rested upon the operation-table on either side of the flanks.

There was a small area of resonance in the epigastrium and in the right flank; the rest of the abdomen was dull. In the mid-line the upper limit of the cyst could be felt and seen standing out vertically above the large pendulous hernia. The patient breathed badly. There were signs of emphysema all over the chest, and both legs and feet were swollen; she was a very obese subject. The skin over the hernia was thickened and eczematous. This was dissected back after a wide circumferential incision had been made. When sufficient integument was raised, the flaps were inverted and clipped with forceps, so that the eczematous surface lay hidden, and the raw, deep aspect of the skin was turned outwards. The abdomen was then opened above the level of the hernia, and a lot of greenish fluid escaped. The incision was carried down to the upper edge of the hernial aperture in the mid-abdominal line, and then continued around the rupture on either side, so as to cut out the sac with its dissected flaps of skin "en masse." A portion of adherent omentum required ligature, as it ran into the sac. The vessels in the omentum were the size of one's little finger, whilst the omentum itself was a dense infiltrated cake, in which nothing typical of fat could be seen. The upper part of the cyst was most intimately adherent to the several abdominal viscera, viz., the coils of small intestine, the cæcum, appendix, ascending, transverse, and descending colons. The parietal peritoneum was enormously thickened and leather-like. There were about two gallons of free fluid in the abdominal cavity. The pedicle of the cyst was small, one inch in diameter; it arose from the right broad ligament. After removal of the cyst and ligature of clamped adhesions, the abdomen was washed out with sterile saline solution, and about one gallon of this fluid was left in. Two broad longitudinal flaps of skin were sliced off from the margins of the abdominal incision, as there was such a redundancy of pendulous parietes, in spite of the fact that the hernia had been dealt with as mentioned above. The walls were then united in layers. The operation took 70 minutes, but the patient rallied well, and eventually made a slow, but apyrexial, convalescence.

PLATE XVIII.

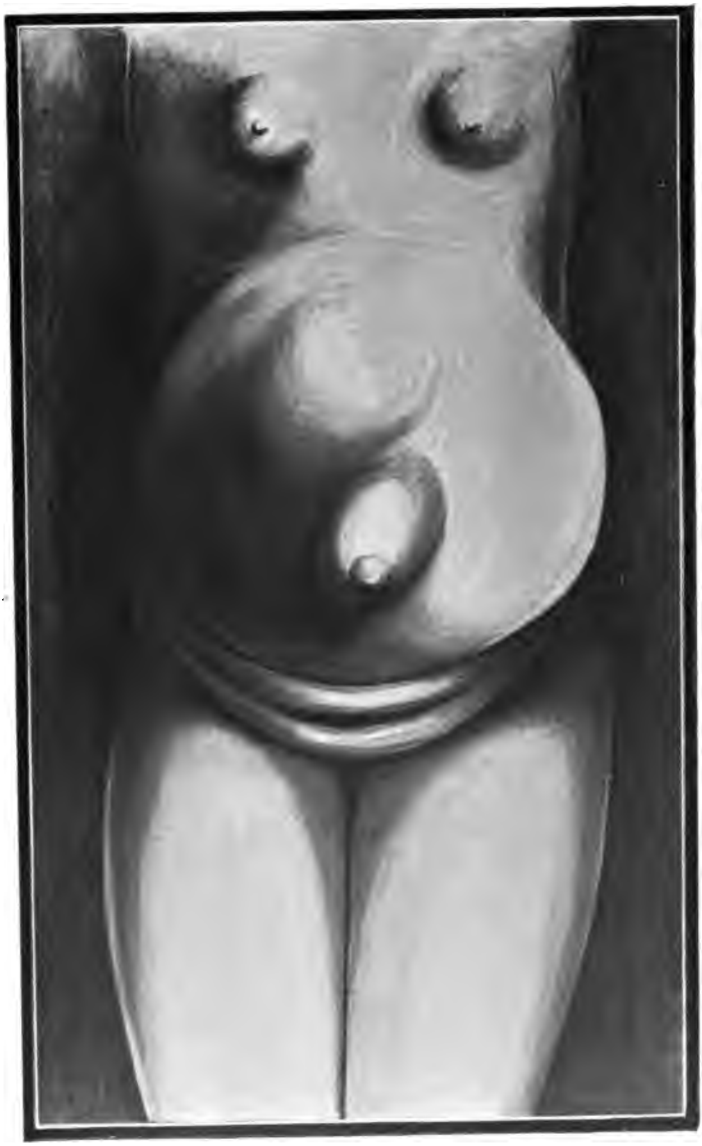

Fig. 4.

The cyst consisted of an upper and lower lobe. The upper had ruptured, and was the one which was tapped. It was covered by yellowish-brown lymph; it measured 28 inches in circumference, and was united by a band of tissue, 11 inches round, to the lower lobe. The upper lobe was unilocular,

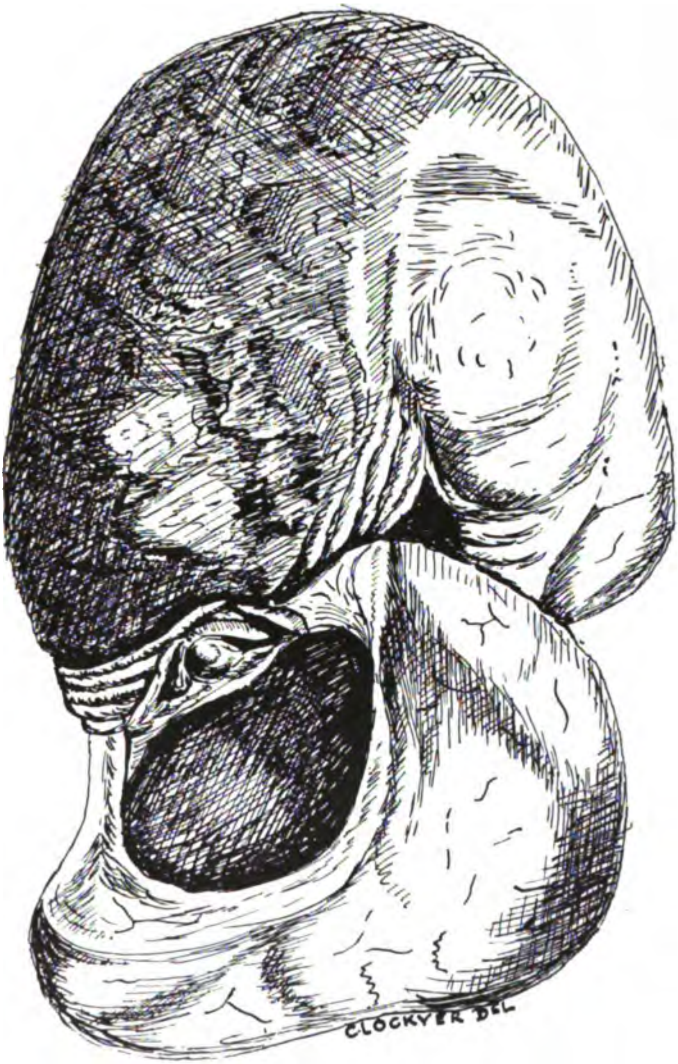

Fig. 5. *Large ovarian cyst, which ruptured subsequent to tapping.*

the lower contained one large loculus, in which were many pedunculated clear cysts, like stalked hydatids. The bulk of this latter lobe was, however, solid, the solid part being of a honeycombed colloid consistence. This lobe measured 22 inches in circumference. It was smooth, no adhesions were attached to it, and it lay in the true and false pelvis whilst the upper occupied the abdomen and pushed up the diaphragm.

The hernia measured 10 inches in circumference, and 4 inches antero-posteriorly. It contained several loculi, and had thick side-walls, but in front it was thin in places.

In this case it is noteworthy that there had been no pain in the abdomen before tapping; from which it is reasonable to infer that the rupture and a good deal of the peritonitis must have supervened after that operation.

(5) *Adhesions.*—Can the presence of adhesions be diagnosed before operation? In a great many cases I think they can. The presence of fibrous adhesions generally causes pain, and the site of the pain corresponds with the position of the adhesions.

The following case bears out this statement:—

Case 5.—Ovarian cyst with strong fibrous adhesions.

L. D., aged 33, had been married 6½ years; she had had no children and no miscarriage. The catamenia began at the age of 12 years, and were always regular, occurring every 5 weeks and lasting 7 days, one towel being used daily. She had had no dysmenorrhœa and no leucorrhœa. Four days ago the menses ceased for 4 months, and the patient noticed her abdomen getting larger from that time; but the menstrual function started again and has since been regular. Pain in the right side of the abdomen and gradual increase in size was what the patient complained of when first seen at Plaistow Hospital.

I found the abdomen uniformly distended, with veins showing though the skin. The swelling reached to the false ribs on either side and to a point 2 inches above the umbilicus in the middle line; it was uniformly dull and tense. A "fluid-thrill" was obtained transversely from flank to flank. The uterus lay at the bottom of Douglas's pouch in a position of retroversion beneath the cyst. At the operation I found a large multilocular cyst proceeding from the right ovary and bound down upon that side by many adhesions; these were tied off and divided, and the cyst removed. The convalescence was uneventful. The pain complained of before operation was, no doubt, due to the adhesions which bound the tumour to the right side of the abdomen and pelvis.

(6) *Burrowing.*—Burrowing may be one of the most troublesome complications with which the surgeon has to contend in performing ovariotomy. It is a great mistake to suppose that cysts in the broad ligament invariably proceed from the parovarium. In 1900 I showed at the Obstetrical Society a multilocular cyst of the oöphoron, which had burrowed into the right mesometrium. Doran has recorded three similar cases; and I have another specimen in my museum, which has not been published. Cystic adenomata of the ovary are, however, fortunately rare in this subserous situation. On the other hand, "hilum cysts" appear not infrequently to burrow. Olshausen, in recording 250 ovariotomies, gives the number of ovarian cysts which lay between the folds of the broad ligament as 46. The greater number of these were "hilum cysts." Still another type of burrowing cyst, which has neither the characters of an ovarian nor of a parovarian cyst, has been met with by Doran. It was unilocular, but its fluid, instead of being clear and non-albuminous, as is the case with thin-walled unilocular parovarian cysts, was highly coagulable. This Doran took to be a dilated lymph-channel. It could not be removed, having no definite wall, but was simply drained; and two years later the patient was free from recurrence. I remember being present and asking Mr. Doran at the operation what kind of cyst he was dealing with, and his reply was: "One of those anomalous retroperitoneal cysts which are met with sometimes."

During the past two years I have had ample opportunity of studying the extent to which cysts may burrow, when once they have invaded the folds of the broad ligament. At an operation for what I diagnosed as a burrowing parovarian cyst, upon exposing the parts by abdominal section, both my colleague, Mr. Angus Kennedy, and myself immediately came to the conclusion the case was inoperable, owing to what we took for a malignant growth of the rectum being adherent to the back of a large broad-ligament cyst of the left side. The abdomen was accordingly closed, the whole procedure taking up but little time. The patient did well for five days; she then suddenly died with symptoms of pulmonary obstruction. Soon after death I opened my operation-wound, and endeavoured to remove the cyst and adherent bowel. This was a matter of great difficulty, and in order to preserve the parts for closer study, I removed the entire pelvic contents. The cyst had stripped up the peritoneum of the left broad ligament in front, laterally, and behind, as far as the brim of the pelvis; it occupied a space beneath the peritoneum, which more than corresponded with its own half of the pelvic cavity. The uterus was pushed bodily to the right. The rectum was bound by cartilage-like fibrous tissue to the back of the cyst. This tissue encircled the rectum like a carcinoma at the upper part of the adhesion, causing a deep diverticulum of the gut just above the obstruction. The bowel had a second adhesion to the cyst lower down. Internally the cyst was intimately blended with the uterus, and in front it occupied the utero-vesical space, raising the peritoneum off the bladder. Below, the left ureter lay embedded in the cyst-wall throughout nearly the whole of its pelvic course. The left round ligament ran on the cyst-wall beneath the peritoneum; and after a most painstaking search by Mr. Targett and myself, we discovered the left Fallopian tube lying curled upon itself (with its fimbriated end looking towards the left uterine cornu) on the front of the cyst-wall. Ovarian tissue was discovered on microscopical examination of the cyst-wall, and owing to the succulent and curdy content of the cyst, I thought it was a tubercular ovarian abscess. This view was supported by the presence of a chain of 6–8 calcareous glands situated along the left ureter, where the latter ran from the trigone to the cyst-wall, and further, because the right ovary was a mere shell of fibrous tissue, investing a calcareous body, the size of a Tangerine orange. I was unable, however, to confirm this diagnosis by the discovery of giant-celled systems in the tubes, ovary, wall of cyst, or glands. The absence of fully-organised structures made the diagnosis of dermoid improbable, so that there is no certainty as to the nature of the cyst, beyond the fact that it was ovarian. I was deeply impressed by the relation of this deep cyst to the ureter; and it influenced me in dealing with a burrowing cyst of the broad ligament upon which, with the assistance of

Mr. Clogg, I operated six weeks ago. The case was one of general pelvic inflammation of long standing. On the right side was a hydrosalpinx coiled around a multilocular cyst which lay in the broad ligament and was inseparable from the right side of the uterus. On the left side there was also a hydrosalpinx, adherent to the upper part of the ovary. The left ovary and tube being densely adherent to the rectum, I removed the uterus and the adnexa above described, after freeing the left ovary and tube from the rectum. The amputation of the uterus was made through the upper part of the cervix. I then discovered an aperture leading into a cyst, which was about the size of a duck's egg, lying between the rectum and cervical stump. The cyst-wall was blended with the rectum by tissue of the density of fibro-cartilage. It extended to the left and caused great depression and bulging of the vaginal fornix on the left of the cervix. Fully convinced that such a cyst, lying so deeply in the connective tissue, beneath the peritoneum in the floor of the pelvis, must have acquired most intimate relations with the ureter, I made no attempt to remove the floor of the cyst-wall, but contented myself with scraping the walls thoroughly and removing a coating of material resembling calves-foot jelly in colour and consistence. I then packed the pelvis with a Mickulicz' iodoform-gauze drain, the last strand of which was removed five days later; and the patient recovered without a bad symptom.

Case 6.—Multilocular ovarian cyst which burrowed between the layer of the left broad ligament and separated the folds of the meso-sigmoid, accompanied by a tubo-ovarian cyst of the opposite side.

F. T., aged 35, was sent to St. Mary's Hospital, Plaistow, in October, 1901, by Dr. Haynes for an abdominal swelling. The patient had been married 9 years; she had had no children and no miscarriages. Menstruation began at 13 years of age, and since the age of 15 it had been regular every 24 days; it lasted 5 days, was never copious until recently, but always painful from the day previous to onset until its termination. The last period continued for 10 days: 20 towels were used, and large clots were passed with much pain in the back and lower abdomen. The patient had for years suffered from leucorrhœa. For the last three years hæmorrhoids had been troublesome, and the bowels were generally constipated. Micturition was normal. Six years before I saw her, the woman had been under the care of Mr. Ward Couzens in hospital at Portsmouth; she was then suffering from a strain, and was kept in bed for two months with fomentations applied to the abdomen and groin. She had a lump in the left groin, for which an operation was suggested, but this was never carried out. Dr. Haynes wrote that he noticed a swelling in the left iliac fossa, the size of a cocoa-nut, on July 28 (about five weeks before admission). This the patient thought had been present for three weeks. She did not think it had anything to do with the lump in the groin complained of six years previously; the latter disappeared entirely under treatment by fomentations. Dr. Haynes found the abdominal swelling was tympanitic and diagnosed a phantom tumour. The distension of the abdomen increased rapidly after July, 1901, and the periods became more painful. On admission general distension of the abdomen was present, this was most marked in the left lumbar region. The superficial veins stood out prominently, especially on the left of the mid-line. The umbilicus was depressed. The abdomen moved well on respiration. By palpation a smooth spherical swelling was felt reaching to a point 2 inches above the umbilicus and lying principally in the left flank. The tumour was uniformly dull except where crossed by the descending colon. No contractions were palpable and auscultatory examination was negative. It was devoid of the stony hardness of a fibroid and of the elasticity of a cyst. Bimanually, it moved with the uterus to which it was adherent. The sound passed 4½ inches. In the left pelvis a solid mass was felt. I diagnosed the presence of a degenerate cystic fibroid of the uterus. At the operation, a pale-coloured cyst presented, reaching to the diaphragm above, and extending outwards into the left flank, and downwards deeply into the pelvis. On its front aspect running from the left uterine cornu outwards and upwards was the Fallopian tube, much stretched and elongated. Behind, the sigmoid was adherent, the tumour having grown into its meso-colon, which had to be opened up in order to separate this part of the tumour from the gut. Large vessels were then seen passing across from the pelvic wall, in the loose cellular tissue, to the base of the tumour, which was solid. The growth had opened up the two layers of the left meso-salpinx, and had a wide pedicle composed of mesometrium, which extended from the side of the uterus to the lateral pelvic wall. This pedicle was ligatured with locked stitches, and the tumour removed. Externally the cyst lay so close to the pelvic brim that its wall had to be cut into, in order to provide enough tissue for the application of a ligature. This outer aspect of the stump was trimmed with scissors after the removal of the tumour, until nothing but the external layer of the wall was left. The contents of the cystic portion of the growth were too mucoid to run through the cannula of Tait's trochar. In the removal of the cyst, adhesions, which bound it to the uterus above and on the left side, had to be carefully divided, and many bleeding points required tying in the left floor of the pelvis. On the right side lay a densely adherent tubo-ovarian cyst, containing greenish-brown fluid like bile. This was the size of a goose's egg (*see* Fig. 6). Attached to it was a subserous inflammatory cyst, the size of a walnut.

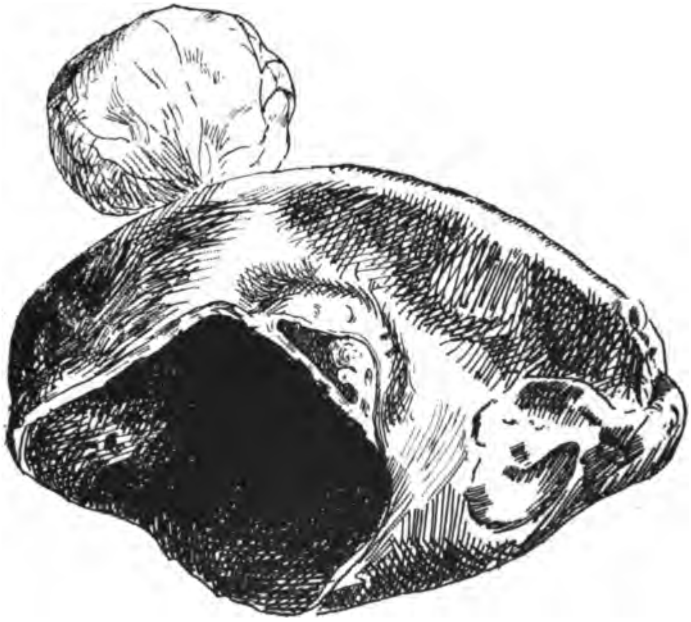

Fig. 6. *Tubo-ovarian cyst accompanying large burrowing multilocular cyst, which lay in the broad ligament and meso-sigmoid.*

Its pedicle was sutured by locked stitches. The peritoneum contained a quantity of serous fluid. The pedicles were covered by peritoneum (Lembert's stitches). The bladder filled during the operation; the pulse never exceeded 80 beats to the minute. The abdomen was sewn up in three layers. Six days after the operation there were signs of general bronchitis; and a pleuritic rub was heard in the mid-axillary line on the right side. The temperature was 101° F., respirations 30 per minute. The wound appeared quite healthy. The patient had to be propped up to relieve respiratory embarrassment. Coughing became very troublesome, the pleuritic rub extended, and signs of pulmonary consolidation at the right base became evident. On December 8 I explored the chest between the seventh and eighth ribs to the mid-axillary line. The fluid withdrawn gave in twelve hours a growth on serum of minute, almost transparent, discrete colonies, which did not increase in size, and which proved on staining by Gram's method to be the Talamon-Fraenkel Diplococcus pneumoniæ. On sitting the patient up, one of the lower stitches gave way, and this was the first indication that the wound was not sound. A troublesome sinus resulted, which gradually opened up communication with the right broad-ligament pedicle. From this in course of time three silk stitches, all locked together, came away. Subsequently the sinus closed completely. The pulmonary symptoms cleared up entirely, and ultimately the patient made a good recovery.

B.—COMPLICATIONS ARISING AFTER OVARIOTOMY.

(1) *Vomiting.*—After the removal of two ovarian cysts and tying off a piece of omentum in the case of a young single woman, the patient was troubled with vomiting for the first three days after the operation. The vomit was yellowish in colour and watery. As the pulse was good, and the temperature normal, and there was no abdominal distension, I was not alarmed, but concluded it was the result of combined shock and anæsthetic (ether).

Sir William Bennett has noted in his operations for the radical cure of hernia that vomiting is more troublesome in cases where he has had to remove extensive portions of omentum; this has not been observed by Mr. Stanley Boyd in similar operations performed by him in exceptional number at Charing Cross Hospital, nor has it come under my own observation in the few cases in which I have had to remove omentum adherent to pelvic growths and gravid tubes. A feature which has been noticed after the removal of omentum in cases at the Charing Cross Hospital is a marked localised tenderness of the abdomen, and this was complained of in my own case, above referred to, but here, owing to the amount of retching, I was not able to satisfy myself that the tenderness was entirely due to plastic peritonitis over the omental stump. As a rule, such sickness as I have described passes off with the free escape of flatus, and to accelerate this I am in the habit of using the rectal tube. I do not employ rectal feeding in ordinary cases, but wait until vomiting ceases before administering nutriment. The very early anæsthetic sickness is best dealt with by sips of hot water. Ice is worse than useless; it creates dryness of the mouth and aggravates the gastric irritability. If vomiting persists for more than 24 hours, hot black coffee, burnt brandy, and bromides may be used; and small doses of white mixture are useful.

(2) *Sinus-Formation.*—This complication of the abdominal wound was a frequent source of annoyance when I commenced abdominal surgery at St. Mary's Hospital, Plaistow, some years ago. This difficulty was overcome by adopting the plan of taking my own sterilizer into the operating theatre, boiling the ligatures myself, and getting the "instrument nurse" to handle them only with a pair of dissecting forceps. A small stitch-abscess may take many weeks in making its appearance. I have known the wound to remain three weeks, or even a month, in an apparently sound condition, and then a small abscess to form about one of the stitches of the buried "running" silk suture, which I use to sew up the anterior layer of the rectal sheath, long after the superficial skin-sutures have been removed. My explanation is that the silk contained pyogenic organisms which were partially crippled, but not destroyed, by attempted asepsis or antiseptics, and which recovered themselves and became virulent in the tissues in course of time. I cannot salve my conscience by a belief in pure mechanical irritation. The reason that silk sutures have given me trouble in the sheath of the rectus muscle and (with one

exception, *see* Case 6) in no other situation, I attribute to the fibrous and feebly-vascular nature of the tendinous material in which the sutures lie. It is a "locus minoris resistentiæ," and organisms will naturally have a better chance of multiplying in such a point of lowered resistance. In one case—already referred to—three locked sutures of coarse silk worked their way out through the abdominal wound along a sinus leading to the right broad-ligament pedicle. It took a year to do this, and I attributed it to a septic ligature being imperfectly covered, first by peritoneum, secondly by the requisite layer of lymph by which it should have become encapsuled. The patient in this instance had her general resistance lowered during convalescence from operation by an attack of pure pneumococcal pneumonia which, it may be assumed, gave any organisms present on a particular strand of silk an extra chance of thriving.

I do not advise opening up long sinuses in order to recover infected ligatures, but these may often be reached and removed by means of a fine crochet-needle. If they are found to be secure, an occasional pull by means of this simple instrument will help to loosen them. They will eventually come away and the sinus close.

(3) *Parotitis.*—This complication after ovariotomy has not occurred in my practice, but I have known it develop after abdominal hysterectomy. This was in a case where one small stitch-abscess formed three weeks after operation. It was a very slight attack of mumps, and occurred nine days after the operation ; it started on the left side, but the right side became affected to a less degree. It lasted a week, and during the attack the temperature ran up to 100·8° F. I am inclined to think the condition was due to a septic condition of the mouth rather than to the slight suppuration in the wound of the abdomen. When parotitis is the result of infection carried by the general blood-stream, it is often fatal.

(4) *Ileus Paralyticus.*—The only experience I have had with ileus has been in two cases which died of infective peritonitis. One case was due to injury of the rectum caused by my removing the wall of an adherent tubercular abscess of the left ovary from the bowel low down, and failing to recognise that I had injured the rectum seriously. I did not

drain or oversew, and, unfortunately, the patient died of peritonitis due to Bacillus coli, with enormously distended abdomen. The other was a case of abdominal hysterectomy for fibroid, in which general peritonitis of streptococcal origin followed immediately, and the patient died in forty-eight hours. I afterwards learned that my assistant had, not long before the operation, dressed a case of erysipelas of the leg, and to this fact I attributed the presence of the streptococci, of which I obtained a culture from the peritoneum. Although I have never had a case of passive distension, dissociated from general peritonitis, I cannot accept Treves' (*Intestinal Obstruction*, p. 280) assertion that "it has no clinical existence." I have seen cases in the Samaritan Hospital in which enormous distension of the abdomen, with excessive vomiting and obstruction to fæces and flatus, was relieved by a purgative draught. I think such cases should be treated by a purge, the use of the flatus-tube, turpentine enemata, and, if need be, by enterotomy.

(5) *Embolism.*—I have incidentally referred to a case in which a patient died with signs of pulmonary embolism five days after an exploratory operation in which nothing was done beyond opening and closing the abdomen. As already stated, no opportunity was afforded me of examining the chest by an autopsy, and so the diagnosis was not confirmed. Cripps mentions having had four deaths from this cause after ovariotomy between the sixth and twelfth day. In my own case, as in those recorded by Cripps, the patient did well, and gave no cause for anxiety, until she was suddenly seized with dyspnœa and pallor, and then died in a few minutes, five days after the exploration. The wound was sound. I opened it, and found no sign of peritonitis. I cannot find any adequate explanation for such an accident. I have only seen one similar case; this was where an operation had been incomplete, and at the autopsy there was no sign of peritoneal inflammation.

(6) *Thrombosis* (*pelvic*).—In the case of double ovariotomy referred to under "Vomiting," just as the patient was about to be allowed to sit up—at the end of the third week—her temperature for the first time ran up to 101° F., and she complained of pain in the left groin. There was tenderness in the region of the left iliac vessels and in Scarpa's space on this side, but no swelling could be felt; nor did the corresponding

limb become œdematous. The symptoms were relieved by prolonging rest for another ten days and applying fomentations over the painful tender area. My diagnosis was "iliac thrombosis." I imagine that a portion of bowel became adherent to the stump of the ovarian pedicle and caused direct venous infection. I have recently seen a similar case, in which convalescence was delayed for three months. The pain was very severe, the leg and thigh had to be kept in a position of fixed flexion in order to give any relief. There was no swelling in the limb, but the pain extended as low as the popliteal space.

(7) *Bronchitis.*—This is not an infrequent complication of abdominal sections, and is due either to exposure or to the irritating effects of ether or to infection carried into the system by the use of a septic inhaler.

If there is any history of previous bronchitis, I avoid the use of ether and substitute chloroform, thereby obviating two of the risks of a further attack, viz., by using a less irritating drug, and administering the same by means of an apparatus which can be made surgically clean. When it occurs, a cardiac cradle must be used, if the breathing becomes hampered.

(8) *Pneumonia.*—This has once complicated the convalescence of a case in which I performed double ovariotomy. I naturally feared that it was an example of "septic pneumonia" from wound-infection, especially as a stitch-abscess was present in the same case. But bacteriological investigation showed a pure culture of the pneumococcus, and it is most improbable that such an organism gained an entrance through the wound. The patient recovered.

TWO LECTURES
ON
THE DIAGNOSIS OF HYSTERIA.[1]

BY PURVES STEWART, M.A., M.D., M.R.C.P.,
Assistant Physician to the Westminster Hospital.

(With Plates XIX.-XXI.)

LECTURE II.

C. MOTOR PHENOMENA.

THESE may be subdivided into irritative and paralytic. Amongst the *irritative phenomena,* the most striking are the so-called hysterical fits or paroxysms. Hysterical fits vary enormously in type and in severity, from a simple emotional outburst of uncontrollable laughing or crying, accompanied perhaps by the hysterical "globus" or "ball in the throat," to the most prolonged, dramatic, violent muscular movements, together with apparent unconsciousness.

One variety of hysterical fit is sometimes mistaken for epilepsy, and there is all the greater liability to make this mistake since the fit is generally over before we reach the patient, and we are then dependent for our information upon the accounts, more or less accurate, of unskilled witnesses. But if the physician is lucky enough to be present during a fit, there is seldom any difficulty in diagnosis. Thus, for example, the patient never hurts herself in falling; there is no stertorous breathing; her face is not livid, nor does she bite her tongue as in epilepsy; but she may bite her lips or snap at the fingers of the bystanders. She never empties the bladder or rectum during the fit; her eyes are generally tightly closed, and if the physician tries to open them, the patient actively resists. A hysterical fit is not followed by coma and hardly ever by vomiting.

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[1] Delivered at the Medical Graduates' College and Polyclinic.

But we must remember that sometimes we have hysterical fits which are post-epileptic, *i.e.*, which immediately succeed an attack of true epilepsy. The antecedent epileptic fit in such cases is usually of the "petit-mal" type, consisting perhaps in a mere transient pallor of the face, with momentary loss of consciousness, and then passing directly into a hysterical fit. Therefore, in every case it is important to enquire very carefully as to the precise mode of onset lest we overlook a case of combined epilepsy and hysteria.

Charcot's "*grande hystérie*," with its initial period simulating epilepsy and its subsequent phases of contortions, kicking and struggling (clownism), passionate attitudes (opisthotonos, crucifixion attitude, &c.), and delirium, often with hallucinations of animals, is not common in this country, but once seen, it is a magnificent performance and can never be forgotten. It is totally unlike any kind of epileptic or organic fit, and its diagnosis is easy. It may last for a quarter of an hour to an hour at a time. This "status hystericus" is commoner than the "status epilepticus," but the patient has none of the stupor which succeeds a severe epileptic fit.

Amongst other varieties of fits, to which I can only briefly refer, we may mention *catalepsy,* in which the patient suddenly becomes speechless, motionless, and stiff. Sometimes she is conscious all through the fit, at other times she is in a dreamy mental state. Meanwhile, if the limbs be passively moved into any posture, however fantastic, they remain fixed there like a doll's limbs. In one case that I saw, the patient, a woman of 28, could be lifted up during the attack by the head and heels and laid across two chairs like a log.

Hysterical trance may come on spontaneously, or may succeed a hysterical paroxysm. It is a condition in which the patient looks as if in a deep sleep; but the muscles are seldom completely relaxed, and we may observe slight tremors of the eyelids. In more severe cases, the heart and respiration may become so feeble and slow that the condition simulates death. Hysterical trance may last hours, days, or weeks; and several cases have been authenticated where such a patient has been buried alive, either deliberately, as in the case of some Indian fakirs, or by accident in this country. Novelists know this, and when the heroine is so buried, she is exhumed in the last chapter by the villain for the sake of a magnificent diamond ring on her finger; whilst her finger is being cut, to get the ring off, the patient awakes. This actually occurred once, I believe, in Scotland.

I can only mention other varieties, such as *somnambulism* and *double consciousness*, this latter being a condition in which the patient's character alternates between normal and abnormal, the two individualities being mutually unconscious of each other, but each one, as its turn comes, taking up the thread where it left off last.

Various *localised spasms* also occur in hysteria. Such, for example, is the large group of "tics" and "habit spasms." A tic differs from an ordinary muscular spasm in being always a psycho-motor act, either an emotional expression or reflex movement which has become a habit when its exciting cause has been removed. Of these, the commonest forms are grimaces, jerkings of the head, trunk, or limbs, and tremors of various parts, rapid or slow. Thus, for example, a lady's maid, æt. 46, had tonic spasm of the orbicularis oculi on both sides (blepharospasm), and could only open her eyes by opening the mouth as well. Another girl, æt. 19, had a clonic or jerking blepharospasm, paroxysmal and associated with "humping up" of one foot. Another girl of 19 had rapid "twiddling" movements of the left thumb and fingers, with pronation-supination movements of the forearm and a pseudo-clonus of the left ankle. Only last week I saw a girl with attacks of pseudo-clonus in both ankles. Another girl of 20, who had suffered from recurrent vulval abscesses, had attacks of rapid antero-posterior movements of the pelvis. Another patient had rapid violent flexion-extension movements of the left elbow whenever a thunderstorm occurred, the hand meanwhile dangling loose at the wrist. Such cases of localised spasms might be multiplied almost *ad infinitum*.

Hysterical Paralysis may be either flaccid or spastic in type, and may affect any of the voluntary muscles; but, unlike paralysis due to organic lesions, it never attacks a single muscle nor the muscles supplied by a single nerve, nor do we ever get the electrical reactions of degeneration. And a further point about hysterical paralysis is that, though it may roughly resemble the posture of an organic paralysis, it never does so quite accurately: there is always some point of difference to be detected. The reason for this I have discussed elsewhere.[1]

Let me show you one example each of hysterical monoplegia, paraplegia, and hemiplegia.

Here is a nurse, æt. 32, with hysterical monoplegia of the right arm, with extreme muscular wasting and claw-hand, of eight months' duration (Fig. 10). This came on after a strain of the shoulder in lifting a heavy patient, and somewhat resembled a lesion of the brachial plexus. But we observed that the trapezius was paralysed, and that the whole scapula was displaced downwards—unlike an organic brachial-plexus case, in which the arm is displaced downwards at the shoulder-joint. Moreover, the electrical reactions were normal, and there was a "glove" of anæsthesia, unlike the "root" anæsthesia of an organic case. The patient was treated for several weeks by battery and massage, without effect at first, but the result proved our diagnosis of hysteria to be correct, for at a religious meeting she was suddenly cured.

Here is a case of hysterical paraplegia, flaccid, of 14 years' duration (Fig. 11), in a woman of 37, which at first sight might be mistaken for a cord-lesion with muscular atrophy and anæsthesia. But the anæsthesia was of the "stocking" type, the electrical reactions were normal, the sphincters were unaffected, and there were no bed-sores; and the result of six weeks' isolation and massage restored the power of walking (*see* Figs. 1 and 2 in previous lecture).

Here is a case of hemiplegia (Figs. 12 and 13) in which the posture was sufficient to diagnose hysteria. Instead of the ordinary posture of an organic case, with the upper extremity flexed and pronated, the lower extremity flexed at the hip, extended at the knee and ankle, and slightly inverted, this patient had a curious posture of the hand and foot unlike organic disease. Moreover, her face entirely escaped in spite of the severe paralysis of the arm and leg; and she had hemianæsthesia and loss of special senses down one side, a combination which never occurs in organic hemiplegia.

The face and tongue are rarely affected in hysterical hemiplegia. But in rare cases we may find spasm of the face and tongue on the affected side, instead of weakness, when the

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[1] *Brit. Med. Journal*, 1902, Vol. II., p. 1407.

PLATE XIX.

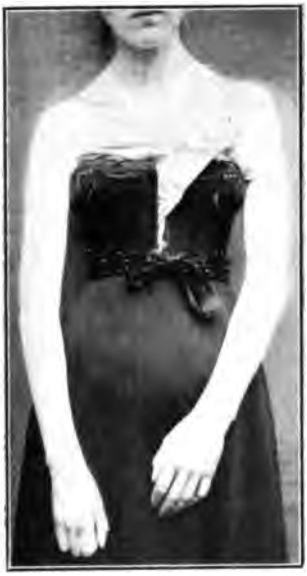

Fig. 10.

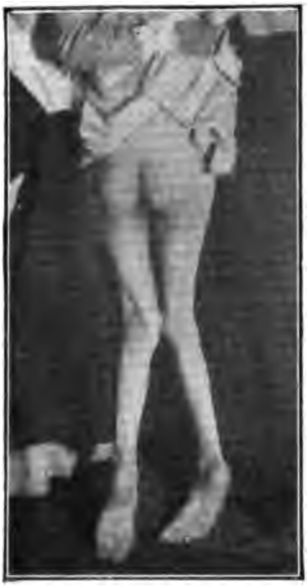

Fig. 11.

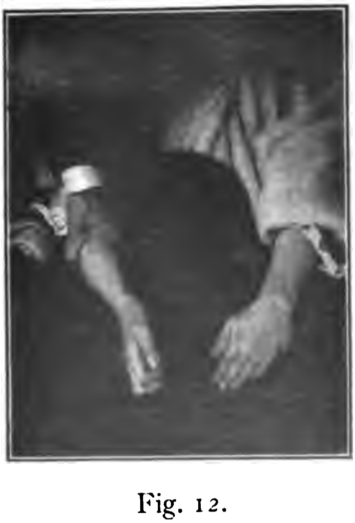

Fig. 12.

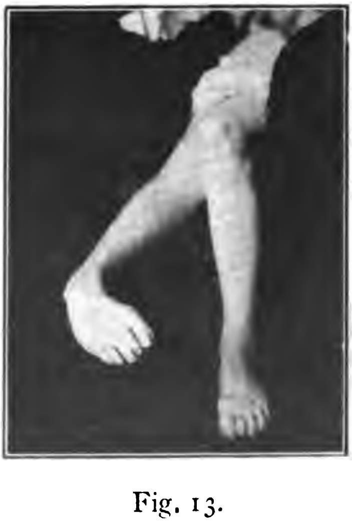

Fig. 13.

patient shows the teeth or protrudes the tongue. This hysterical *glosso-labial hemi-spasm* is exceedingly rare, but I am able to show you a good example (Fig. 14).

I may also refer to "Lasègue's symptom," in which a patient with an anæsthetic limb cannot move it when the eyes are closed, but is able to move it when she opens her eyes and looks at it. Another feature worthy of mention is hysterical pseudo-ptosis. In organic ptosis due to lesion of the 3rd cranial nerve, there is always a compensatory over-action of the frontalis muscle. But in hysterical pseudo-ptosis this is absent, and the condition is really due, not to paralysis of the levator palpebræ, but to spasm of the orbicularis oculi.

Before leaving the motor phenomena of hysteria, it may be well to refer to the gaits of hysterical patients, which are sometimes most peculiar. Thus one patient, a doctor of 55, used to flourish his left leg in the air and bring it down with a stamp like a unilateral locomotor ataxia. The phenomenon was so dramatic that he had to carry a stick to beat off the crowd of little boys who used to study his gait in the streets.

Astasia-abasia is a hysterical condition in which, although the patient can move his legs normally when lying or sitting, he collapses at once when he tries to stand or walk. A boy, aged 12, had this symptom for a long time, but was cured by an application of the wire-brush to his spine. Another patient whom I saw was a worthy married lady who, every now and then, when walking, sat down suddenly on the ground, rolled backwards and spread out her legs like the letter V. By appropriate treatment she, too, was cured of this distressing symptom.

Perhaps the commonest hysterical gait is a dragging gait, in which the patient trails the limb helplessly along, often scraping the inner border, or even the dorsum of the foot, on the ground, unlike an organic hemiplegia in which the outer side of the sole is dragged.

It has been stated that muscular atrophy does not occur in a hysterical limb. But this is not quite accurate, for in certain rare cases, one of which is shown in Fig. 10, we may meet with advanced atrophy. But this atrophy is simply due to disuse and is not associated with the electrical reactions of degeneration.

Contractures of the most pronounced type may be met with in hysterical paralysis. But here, again, they always differ in some respect from organic cases. I show you the photograph (Fig. 15) of a soldier, aged 32, whose horse rolled on him at the Tugela, and who afterwards developed a stiff arm, flexed at the elbow and wrist, and with the thumb and index held stiffly parallel.

A study of the reflexes is of great diagnostic importance in every case of hysteria. The deep reflexes may be normal or exaggerated, but the knee-jerks are never lost in pure hysteria. True ankle-clonus does not occur, but a pseudo ankle-clonus is often met with. In one girl of 19, the subject of thread-worms and pruritus ani, this pseudo-ankle-clonus used to come on spontaneously when sitting or standing loosely. One can often distinguish it from a true organic clonus by a peculiar upward start of the foot before it sets off on its first downward push. Also pseudo-clonus is poorly sustained as a rule.

The superficial reflexes are often diminished or absent, especially on the anæsthetic side. The plantar reflex in hysteria, if present, is always of the normal flexor type, never of the extensor or Babinski type. The extensor plantar reflex practically only occurs in cases of disease of the pyramidal tracts and in infants who have not learned to walk and in whom the pyramidal tracts are not yet developed.

The pupil-reflex to light is never lost in pure hysteria. I remember one case of fixed dilated pupils in a hysterical woman, but this was due to the taking of belladonna by the patient herself.

As to the bladder and rectum, although we may have frequency of micturition in hysteria, we never have true dribbling. Retention of urine, on the other hand, is a fairly common symptom. It once broke out as an acute epidemic in a school for young ladies where the visiting doctor was a very young man, and it continued until he judiciously handed on the duty of catherisation to a female nurse of mature years. The symptom at once subsided in a gratifying manner.

D. VISCERAL AND VASOMOTOR PHENOMENA.

It is important to remember that hysteria affects the sympathetic nervous system as well as the cerebro-spinal.

PLATE XX.

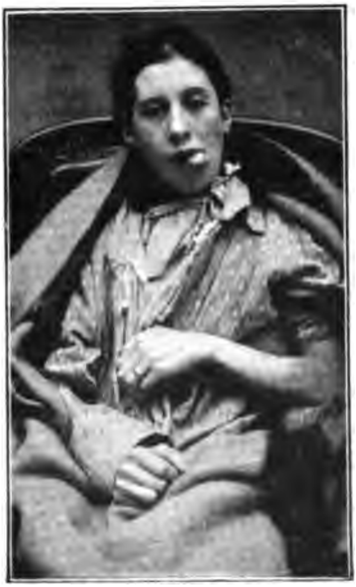

Fig. 14.

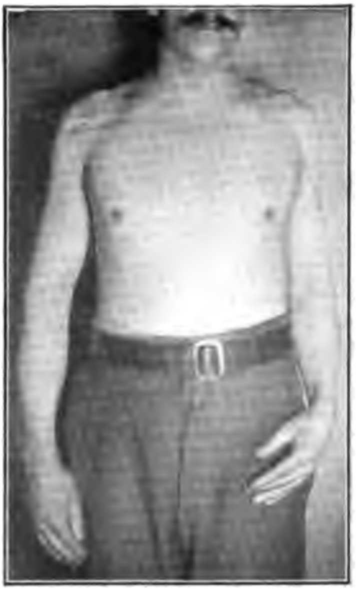

Fig. 15.

Time, however, does not permit me to do more than refer very briefly to some of the visceral and vascular phenomena.

We may meet either with abnormal slowness of the heart or abnormal rapidity with palpitation, chiefly paroxysmal and constituting a variety of pseudo-angina, especially in hysterical young mothers who have been lactating too long. These cases are easily distinguished from true angina by the absence of signs of organic cardio-vascular disease.

In the digestive system we meet with curious hysterical phenomena. Aërophagy, or swallowing of air, is achieved chiefly by gulping movements of the pharynx. I remember a little school-boy who could swallow air and distend his abdomen till his waistcoat could not be buttoned over it. We are all familiar with hysterical dysphagia or spasm of the œsophagus, with its sudden intermissions and the difficulty experienced equally with liquids and with solids, yet in which a large stomach-tube can easily be passed through. The hysterical "globus," or "ball in the throat," which the patient tries to swallow, is an emotional phenomenon often met with at the onset of a hysterical paroxysm. Hysterical vomiting has always to be excluded in gastric disorders of young women. It is often associated with *anorexia nervosa*, where the patient will take hardly any food. "Fasting girls," of whom we now and then read in the daily press, are generally examples of this kind of hysteria. And they may be extraordinarily emaciated, but even they do take a little food now and then.

Rhythmic movements of the stomach or intestine with curious rumbling noises are sometimes met with. The commonest is the intestinal variety, and one sometimes hears these noises at dinner-parties in nervous young servant-maids waiting at table. Much less frequently we hear violent gastric borborygmi. One young girl whom I saw had constant, noisy to-and-fro gurgling in the upper part of the abdomen, like a steam-pump, and on palpating the abdomen the stomach could be felt rhythmically contracting and relaxing, flowing and sucking air backwards and forwards through the pylorus. This phenomenon was so startling to strangers that the unfortunate girl had to retire to her own room, if friends came to call on the family.

The French patient who earned an honest living at a certain music hall in Paris by making musical noises with his anus, was probably also an example of visceral hysteria.

I must also remind you of the pseudo-pregnancies which now and then occur, in which the spurious enlargement of the abdomen sometimes goes on to a spurious labour. Then "parturiunt montes, nascitur ridiculus mus"—all that is produced is, at the most, a small uterine cast. Phantom abdominal tumours can best be differentiated from genuine ovarian or uterine enlargements by giving an anæsthetic, when the abdomen at once collapses.

It is sometimes more difficult to diagnose pseudo-appendicitis, Thus one patient whom I saw, aged 33, had had her abdomen opened twice in different London hospitals for supposed appendicitis—recurrent pain and tenderness in the right iliac fossa, with constipation and vomiting. But she also had right-sided hemianæsthesia, with loss of special senses all down that side. One was thus led to suspect the hysterical nature of the abdominal symptoms, and accordingly her next attack was cured by sal volatile, without laparotomy, and she has had none since.

Hysterical diarrhœa sometimes occurs, as in the case of a public speaker who was often attacked in this awkward way just when his turn arrived to address the audience.

Spontaneous hæmorrhages are very rare in hysteria and should never be diagnosed as hysterical unless all other causes can be excluded. But a certain number of cases of pseudo-hæmoptysis and pseudo-hæmatemesis occur. In one girl whom I watched, the phenomenon seemed to be due to sucking of the gums; in another it was apparently the result of pharyngeal suction. In both cases, the most experienced physicians who saw the case in consultation failed to discover any organic cause in the chest or abdomen.

Sometimes a limb affected by hysterical paralysis and anæsthesia may show abnormal vasomotor spasm, so that if pricked or cut it bleeds less freely than normal.

Secretory phenomena also occur, though rarely, as for example in blood-stained tears or blood-stained sweat, sometimes unilateral. Polyuria often occurs after a hysterical fit, whilst hysterical anuria or suppression of urine is extremely uncommon.

PLATE XXI.

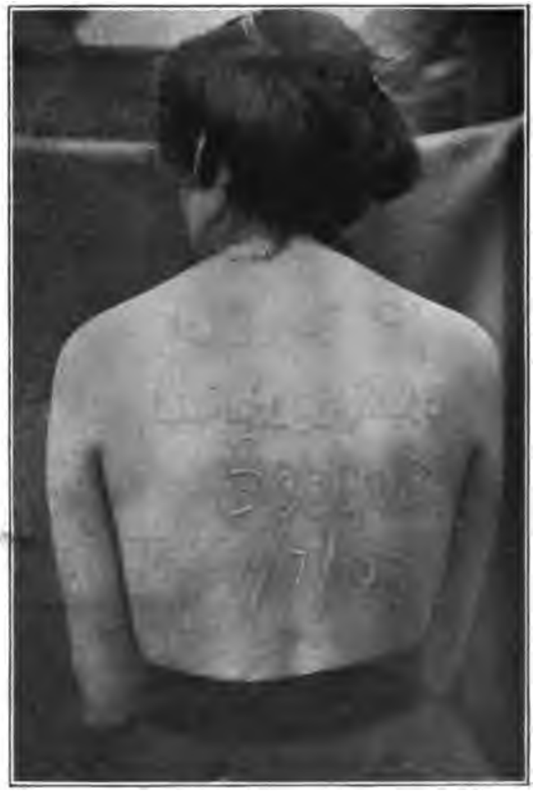

Fig. 16.

Case of "urticaria scripta" in a hysterical patient.

Certain skin-affections occur in hysteria. Cutaneous hæmorrhages are rare, if we exclude cases of voluntary traumatism. The gangrenous patches described as hysterical gangrene are generally self-inflicted, *e.g.*, by means of caustics or other methods. Hysterical blue œdema sometimes occurs, especially in contractured limbs. It usually affects the skin over a joint and produces a degree of cyanosis and swelling, but this swelling does not pit on pressure. Dermographism is commoner in hysterics than in normal people. I show you an excellent example in a girl with hysterical tremor of the legs in whom, when the skin is stroked with the finger-nail, a white raised wheal appears and remains for an hour or more (Fig. 16). This condition is sometimes called factitious urticaria or "urticaria scripta." It does not itch, unlike ordinary urticaria.

Hysterical cough is very common. It is usually loud and hacking, going on all day and ceasing during sleep. It is not accompanied by expectoration, and is commonest in young hysterics. We may have other peculiar modifications of respiration. Thus in a girl of 24 who had hysterical fits, the expiration was a curious grunting noise of a bigeminal type—two grunts between each inspiration. We also meet with paroxysmal sneezing, hiccup, and yawning. One girl of 10 with hysterical hemianæsthesia yawned persistently for three weeks during her waking hours. She then stopped and had an attack of hysterical mutism lasting for two months.

Such are some of the chief diagnostic phenomena of hysteria. But there are many others to which I have not had time to refer. The diagnosis between hysteria and organic disease may be easy or difficult, and in doubtful cases I would suggest that special attention be paid not only to the psychical symptoms, but to the special senses, the condition of the optic discs, the presence of a hysterical type of anæsthesia, the posture in motor paralysis, and the condition of the reflexes, especially the plantar reflex and the bladder.

And we should never forget that hysteria and organic disease may coëxist in the same case.

PHTHISIS IN RELATION TO LIFE-ASSURANCE.[1]

By T. D. LISTER, M.D., M.R.C.P. Lond.,

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At a time when insurance offices are talking of eliminating the medical examiner altogether, it would seem, perhaps, a superfluous task to speak of the principles which govern the relation of phthisis to life-assurance. But such a reform—if it be a reform, and not merely a new way of drawing a shy section of the public into the insurance fold - will take some time yet to effect, and its oncoming will not be hastened among the class who do not mind examination by the fact that those who elect to insure under the new method have to pay an additional premium for the first year or two—that is to say, that their executors only get half or a third of the nominal value of the policy, if death takes place so early as within twelve months, and only a proportion, if within two years. For this reason there may be still some time before we cease altogether to be asked to undertake the examination of proposers.

The only excuse that I can offer for my selection of this subject for to-day's consideration is the fact that the modern popular substitution of hope for the ancient attitude of despair in regard to consumption is having, and will continue to have, a far-reaching influence on the manner in which offices deal with the disease. Public opinion has a good deal to do with insurance. Since the insurance of under-average lives has developed so as to become a considerable proportion of insurance business, and the fact is recognised that it can be undertaken at rates which, although higher than normal, are always, in all insurable lives, regulated fairly by competition between the offices, the public takes care—at any rate the intelligent, educated class of insurers—that such a well-known fact as the infectivity of phthisis should be allowed for. Often a proposer will endeavour to explain the occurrence of phthisis in his family by a story of accidental infection in a way which he believes is favourable to the moderation of his premium, and, if the result be unsatisfactory in his view, he will take his proposal elsewhere. It is therefore necessary, when examining for any particular office, to exercise a very even judgment in such instances. A too unfavourable report, leading to a heavy addition, and followed by acceptance at another office on somewhat better terms, is to be as carefully avoided as a too favourable report leading to acceptance at average rates and followed by a heavy loss. Either course is liable to lead to a loss of confidence on the part of the office. It has sometimes seemed to me that we do not always realise our responsibility in this matter. For the medical examiner exercises his skill in a twofold capacity, which has no relation to the interests of the individual applicant, but only to the office. He is employed by the office to safeguard existing policy-holders both by excluding bad lives, and by including all lives insurable at fair rates. In a lecture by Dr. Pye-Smith, in 1897, this duty was expressed in a nutshell in the words, " Reject all you must, accept all you can."

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[1] A Post-Graduate Lecture delivered at the Mount Vernon Hospital for Consumption on July 2, 1903.

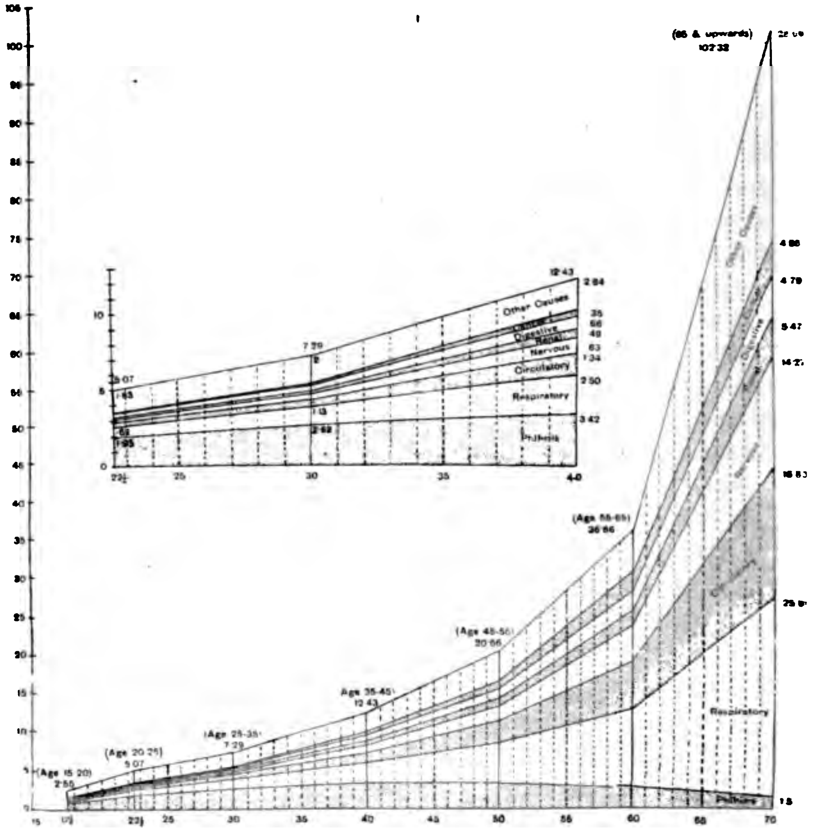
As the result of medical examination and the careful selection of lives, the number of insured lives that die is less than that of the whole population. Among the whole mass of insured lives there are certain classes that do not have an average chance of survival, and those that die of phthisis form the largest number of these.

Of 1,000 persons living at any age a certain number will die at that age, and this proportion of deaths is the risk that the office has to face in insuring them for one year at that age. It is of course made up of several groups of diseases, and is readily analysed at any age-period by the use of figures given in the Registrar-General's returns. I would ask you to look for a moment at a diagram made for a paper read at the Life-Assurance Medical Officers' Association in 1901.

The ages are indicated in the base-line. The vertical lines indicate according to their length the number of deaths per 1,000 at each age. You will notice that the figures given in the Registrar-General's returns do not allow of a large number of calculations, and the vertical lines are exactly calculated only for the mean ages between 15 and 20, 20 and 25, 25 and 35, and so on. You will see that at the age of 15-20 (mean age 17½) the proportion of deaths is 2·55 per 1,000; at 22½,

The Constitution of the Normal Risk in Occupied Males.
400,057 deaths.

The risk at any age is built up of parts as shown.



From the *Transactions of the Life-Assurance Medical Officers' Association*, 1902: "On a Graphic Expression of Extra Risk," by T. D. Lister. It is to be noted that this table is not derived from insured lives.[1]

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[1] For the loan of the blocks for this and the following illustrations we have to thank the Life-Assurance Medical Officers' Association.

5·07 per 1,000; at 30, 7·29 per 1,000; rising to 102·32 per 1,000 at 70, which is taken as the mean age of lives aged 65 and upwards. The line joining the tops of these vertical lines makes a rough curve rising at first slowly and later rapidly above the base-line.

Now between the base-line and this curve are certain spaces alternately light and dark. I will only draw your especial attention to the bottom one, for it represents the proportion of deaths due to phthisis at every age. The other spaces represent the other main groups of causes of death, and were each calculated for every age-period. You will notice that they differ most remarkably in shape from that of phthisis. For the greater part of the diagram the area which marks the deaths from phthisis is practically a band of equal width throughout, while that of every other important group of death-causes begins as a minute wedge and grows constantly broader from youth to old age.

In other words, as has often been pointed out, almost as many deaths from consumption occur in the later years of life as in the earlier years, per 1,000 lives at that age, and the number is almost constant at all ages.

The diagram seems to show a falling off in the width of the phthisis-band in the last few years; but when we remember how often tuberculosis may be the termination of old-standing chronic bronchitis and emphysema, and be unrecognised on account of the previous condition of the lungs, there is no doubt that some at least of this falling off is due to the non-recognition of tubercular infection in an already-damaged lung. When old people with bronchitis seem to become more seriously ill, an examination of their sputum may show that infection by tubercle has been added to their other troubles.

For the purposes of this lecture it may be assumed that phthisis is an equally frequent cause of death at all ages during which insurance is usually effected, say, from 30 to 60. Of course, as age increases, right away from the beginning, other causes of death than phthisis begin to assert themselves with an increasing frequency, which is illustrated by the constantly broadening shape that they each give rise to in the diagram. Hence, since phthisis does not increase, the percentage of deaths due to phthisis as compared with the total number becomes gradually less. Looking at the little diagram which represents on an enlarged scale the causes of death between age 22½ and age 40, you will see that at the younger age 1·95 out of 5·07 deaths are due to phthisis; at 30, 2·62 out of 7·29; and at 40, 3·42 out of 12·43. These figures represent 38 per cent., 36 per cent., and 27 per cent. respectively. In old age, without allowing for the possible error due to the suggested non-certification of senile pulmonary tuberculosis as phthisis, which would probably quite double the figure, the percentage, according to the figures given in the diagram, has fallen to under 1·5 per cent. (1·5 in 102·32).

We must regard phthisis, therefore, as causing an approximately equal number of deaths at all ages, though the percentage as compared with other causes of death constantly falls as age increases. If the phthisis-risk be supposed to be increased in any proposer, it follows that the addition made in youth must be greater than that in age.

Let us ask ourselves what we mean when we say that a proposer has an increased liability to phthisis from an insurance point of view. Of every 1,000 occupied males at age 30—that is, including all occupations and localities—2·62 die of phthisis. In some occupations more, and in some less, than this number die at this age of phthisis. For instance, among drapers and warehousemen, 4·4 per 1,000; among clerks of commercial houses, 3·48 per 1,000; among agriculturists, 1·65 per 1,000; and among clergymen, 1·56 per 1,000 die at 30 years of age from consumption.

One could say with truth that drapers and commercial clerks have an excessive liability to consumption as compared with the whole mass of occupied males. But no one would think of recommending that an extra premium be charged to people in those two occupations because of this increased liability as a class, any more than of recommending that a premium below the ordinary be charged to clergymen and agriculturists because of their diminished liability to the disease. And this holds good, although we can see from the figures that the increased liability to consumption is the only cause of the difference between the mortality of drapers or clerks, and that of all occupied males, and a more than sufficient one.

I have put them in a table.

Deaths per 1,000 at Age 30.

Class.	Total Deaths.	Deaths from Phthisis.	Deaths from other Causes.
Occupied males - -	7·29	2·62	4·67
Drapers, &c. - - -	8·52	4·4	4·12
Clerks, &c. - - -	7·73	3·48	4·25
Agriculturists - - -	4·83	1·65	3·18
Clergymen - - -	4·23	1·56	2·67

The classes given in this table would all be insurable at average rates, if the individuals be fairly representative members of their own class; and average clergymen and average drapers pay the same rate, although the mortality of one class is only half that of the other, as a whole. In other words, there is a wide margin for average or so-called "first-class" lives, and this is true at all ages and, I may add, of all diseases. It is to be remembered. however, that insurance tends to bring the extremes together, and the insured draper has, no doubt, a little better life than that of drapers as a whole, while the insured clergyman has equally a little worse life than that of clergymen as a whole, and the margin is not really so wide in practice as it appears from a table of unselected lives.

We should not, I say, advise an addition to the premium in the case of drapers merely because their class-liability to phthisis is nearly 75 per cent. greater than that of all occupied males, and three times as great as that of clergymen.

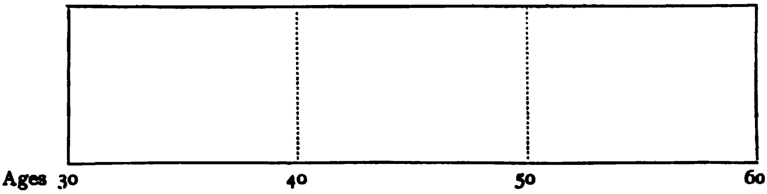
We have to go a little further, and in deciding to put a life in the under-average class we have to consider whether the individual himself has a greater liability to phthisis than the average of people of his own class in the same circumstances and under similar environment. Afterwards, when deciding what addition should be made, it may be advisable to consider the special danger or safeguards of his individual circumstances and environment.

The extra danger may be supposed to be increased or diminished according to the nature of these. For instance, the addition to be made for a suspected tendency to phthisis in a draper would often be greater than that required in a clergyman or agriculturist, the lives otherwise corresponding.

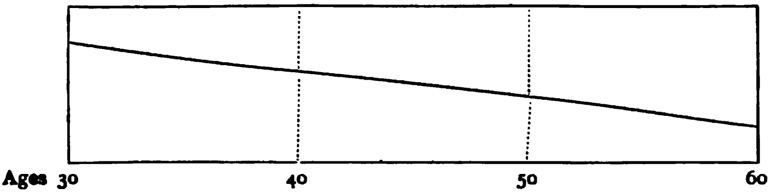
Let us go back for a moment to the conception that the normal danger from phthisis is the same at all the insuring ages, being represented in our diagram as a band of practically equal width throughout. I would ask you to join with me in a few considerations as to the constitution of that band.

I pointed out how the representation of the phthisis-part of the normal risk of insurance differed in shape from that of all the other groups of diseases. The latter begin as very small parts of the total risk, phthisis being equal to them all put together, but they each increase rapidly, and cause the rise of the mortality-rate as age increases, phthisis taking no part in the production of this rise. The phthisis-risk is represented by a band of equal width at all the usual insuring ages.

But it is possible to imagine that the phthisis-risk is not simply to be represented by a homogeneous band. Let us represent the phthisis band between ages 30 and 60 as an elongated rectangular figure.

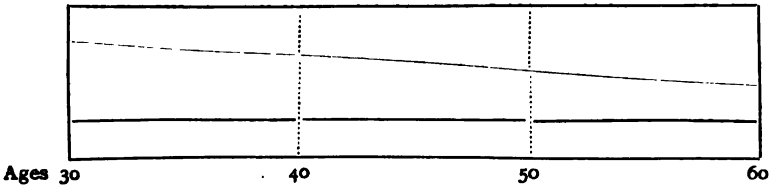


A band such as this may be homogeneous, or it may be cut up in three possible ways (and only three), if each of its parts maintains a regular tendency from beginining to end of the band. It may be homogeneous throughout, as in this diagram, or it may be divided into two parts, an increasing and a decreasing part, where a sloping line divides it into



two parts, one which increases as age advances, and one which decreases.

The third way, to which I wish to call your special attention, is that it may be made up of three parts, an increasing part, a decreasing part, and a constant part.



Now looking at this third diagram, I would remind you of our customary clinical recognition of the three dangers which give rise to tuberculosis, viz., hereditary vulnerability, acquired vulnerability, and the constantly-present danger of infection. May we not suppose that the band representing the normal risk of phthisis is thus built up, and that the total risk is constant because a certain number of people inherit vulnerability, a certain number acquire it, and the risk of infection is always about the same? May we not suppose that those who inherit vulnerability as time goes on become less affected by the constitution they derived from their forbears, and more by their own circumstances and environment, the inherited vulnerability thus passing gradually away? Finally, may we not suppose that among those free from inherited taint, there are some with whom unfavourable surroundings and circumstances, and damaged physique, cause gradual loss of any resistance they acquired from their forbears, acquired vulnerability thus gradually increasing? The rate at which individuals lose inherited characteristics and develop their own can scarcely vary, and so a fairly even balance is maintained between the amount of decrease of the one and the amount of increase of the other among the whole phthisical population which is thus represented. For the purposes of a broad illustration of the composition of the normal risk from phthisis, I ask you, therefore, to think of it in a graphic and fanciful way as being represented by the third diagram. That is to say, it is made up of a decreasing part, corresponding with inherited vulnerability; an increasing part, corresponding with acquired vulnerability, and a constant part, corresponding with the ever-present risk of infection.

We can suppose any or all of these to be larger than normal, in a proposer coming before us, and to require an addition to the premium.

We asked ourselves what is the meaning, from an insurance point of view, of an increased liability to phthisis, and I think we can say that it is an excessive danger from either of the three factors—inherited vulnerability, acquired vulnerability, or infection, or from any two or all three of these together.

When we examine a proposer for life-insurance, so far as phthisis is concerned, we may, therefore, consider the matter in these varying aspects. Firstly, as regards inherited vulnerability. In the first place, a family history of phthisis does not always imply an inherited vulnerability. A family history of phthisis is made up of infection and vulnerability. The latter is quite a different thing from family infection, and I would ask you to keep the two very clearly differentiated when filling up your insurance paper. For this purpose, when we are gathering the family history of the proposer and phthisis appears as a cause of death among the relatives, it is well to enquire fully into the circumstances preceding and accompanying its occurrence. In cases of doubt as to the nature of the disease an enquiry as to its duration often affords a clue. Deaths from chest-disease under 30 are mostly phthisical, as my first diagram showed. Nowadays, owing to the spread of knowledge as to the nature of this disease, a proposer, as I said, will very frequently volunteer an explanation of how it happened that a particular member of the family died of phthisis. This explanation may or may not have any favourable bearing on his case, but an example of the explanation that would not be unfavourable to the healthy, robust proposer would be that a brother who had "gone to the bad" and taken to drink and dissipation in a foreign town, had finally died of phthisis under conditions of privation, after long separation from his otherwise healthy family. But you will find every degree of family invasion by tuberculosis. Rules of the most elaborate kind were formerly made to meet the various degrees of inheritance, various additions being advised according to the number of relatives affected, and the age of the proposer. But if we reflect that a family history of phthisis must begin in the infection of the first case in the family, we can see that the circumstances under which other cases arise are of great importance. Family life predisposes to infection, and several cases may develop in a family not really possessing a marked vulnerability. This results from infection by the excessive and cumulative dose of the virus that will be likely to fall on any one of those who dwell intimately together with a bad case. Such are real cases of family infection. It is very frequently seen that two or three members of the family die of phthisis within a year or two of each other, for instance, a mother and the two daughters who nursed her, or a son and the mother who nursed him. Such cases when properly investigated often mean family infection far more than vulnerability. A robust proposer in good circumstances, long separated from any of the major risks of infection arising from co-dwelling with his infected family, may safely be taken at the ordinary rate. But it must never be forgotten that the class who have a family history of phthisis have an excessive mortality on the whole, and careful investigation is required before making exceptions.

Before leaving the question of inheritance, I would like to be allowed to suggest another way of looking at it. If I may use the parallelogram of forces as an illustration of heredity, and represent a life as a resultant force, its component forces being derived from the parental lines, I think we can imagine, by carrying the illustration further back, that every one of our individual lives may be looked upon as the final resultant force of a whole series of forces "resolvable," as we used to say in mechanics, into pairs of ancestral components. The origin of each life starts at a point in time, and may be figuratively considered as being the beginning of a new force having a certain inevitable magnitude and direction. Its components are themselves each resolvable into remote components and so on.

Deriving an impulse from each component it partakes more of the magnitude and direction of one or other according to circumstances, which may be purely accidental. But we may regard its magnitude as being comparable to vitality, and the direction as the line of pathological tendency.

From such a simple illustration it may be still further surmised that, although the new life may, and probably will, resemble one or other of its immediate predecessors most closely, yet it may also resemble some remote ancestor instead, by reproducing some particular antecedent association of magnitude and direction.

Returning to the "family history of phthisis" with some such ideas in mind, we may argue that any member of a family may exhibit tendencies to robust health derived from a sturdy old ancestor who existed before the family history of phthisis began. And I would like to add, as an interjected opinion, that a member of an apparently healthy family, so far as its recent members are concerned, may also possibly derive a vulnerability to consumption from an ancestor of delicate fibre. This is to adopt a rather fatalistic view of inheritance, but one which will afford some speculative interest and amusement for those who are of a contemplative mood. I think we must consider heredity to play an important part in the production of phthisis, not so great as was supposed before the days of the establishment of its infective nature, yet not so little as some would lead us to believe. The personal condition of the proposer is very important, and, whatever its nature, may, as I have suggested, be regarded as being itself largely due to heredity, if one wishes to think so. What we have to consider, therefore, in relation to the hereditary vulnerability to phthisis, is whether the personal condition of the individual corresponds with that of hereditary resistance or predisposition.

In certain offices a question is asked which helps to shed light on doubtful cases, and that is as to the degree of resemblance of the proposers to one or other of the parents. If one of these have died of phthisis, and the proposer is said to resemble that one, a certain amount of hereditary vulnerability may be inferred. Moreover, it has been shown, without a shadow of a doubt, that a family history of phthisis in a proposer under the standard weight for height and age has a very important bearing on insurance.

The relation of the proposer's weight to his family history as a factor in estimating his phthisis-risk has been illustrated in a paper issued by Dr. Marsh, of New York, in 1895, and his results are summarised in the following table :—

TABLE showing the PERCENTAGE of DEATHS from CONSUMPTION, out of the total number of Deaths at the various Ages, of Proposers under various conditions of Family History and Weight, separately and combined. (From figures given by Dr. Marsh of the Mutual Life-Insurance Company of New York, in a Paper published in 1895.)

	A.		B.		C.		D.	
Age.	Family History.		Weight, all Classes.		Over Weight.		Under Weight.	
	Untainted.	Tainted.	Over.	Under.	Untainted Family.	Tainted Family.	Untainted Family.	Tainted Family.
30	28·5	37·6	20·0	44·1	17·8	25·0	41·1	48·9
30-39	13·3	20·2	9·6	23·2	8·5	11·3	19·1	28·2
40-49	6·8	9·2	5·5	12·1	4·6	6·7	10·7	16·8
50 and over.	4·6	4·9	3·0	8·3	2·7	3·5	9·3	7·3

These figures afford us a good deal of information. Firstly, column A. shows that a family history alone increases the probability of phthisis among proposers, this influence diminishing as age advances. Secondly, column B. shows that light-weights, irrespective of family history, have a greater liability to phthisis than those who are above the average, this influence remaining much the same at all ages as regards the proportionate difference between the light and heavy classes; that is to say, rather more than twice as many deaths from consumption occur among all age-groups of light-weights as among heavy-weights. Thirdly, column C. shows that among heavy-weights there is less consumption at each age, whether the family history be good or not, than there is among either of the corresponding groups in column A.; that is to say, that a good weight-record discounts a bad family history, speaking broadly, and is a favourable feature in any individual case, as regards the phthisis-risk. But I would remind you that these figures deal with the percentages of all deaths due to phthisis, and for each figure there is the remaining number per cent. to make up the 100. A lower phthisis-figure in this table means that the particular group died more frequently of other diseases, not that their total mortality was affected. That is not given, and would of course differ in the various groups. Fourthly, column D. shows that among light-weights there is more consumption at each age, whether the family history be good or not, and that a tainted family history in those of light weight is of the greatest significance of all. These figures suggest the manner in which we should deal with cases of family history of phthisis.

A greater addition is required for family history in a younger life than an older one, as I have already said, of all phthisis-risks. Indeed, as a general rule, this is true, but in a less marked degree, of other diseases also; but I need not refer to that to-day. The influence of family history, as Dr. Pollock has expressed it in the Medical Handbook of Life-Assurance, "strikes in early life, is much diminished after 45, and is probably not extinguished till 70."

Allowance may be made for robust personal health, which frequently, but not always, renders an addition for the phthisis-risk unnecessary. But a very considerable addition must be made for the phthisis-risk when the proposer with a tainted family history is below the average weight.

Lastly, I would ask you, whenever possible, to make such enquiries as will enable you to form some opinion as to whether the family history, if at all marked, is due to hereditary vulnerability or family infection. We must always remember that an average resistance may be overcome by bad environment and an excessive dose of bacilli. A group of cases occurring in a family within a very few years of each other under conditions which render infection probable is suggestive of infection rather than vulnerability.

It is a curious thing, in the light of our modern views, that the offices do not ask whether the proposer dwells with those who suffer from phthisis, though it might be a very useful question to ask on one's own initiative.

What I have to say in regard to the other two kinds of extra risk from phthisis, viz., the increasing and constant kinds, will of necessity be very brief. They arise from conditions with which everyday clinical experience render us all familiar.

An excessive danger from phthisis, increasing as age advances, may be supposed to exist where an unhealthy environment acts for a long time, or where there have been repeated attacks of any pulmonary complaint, especially if to either be added the excessive use of alcohol. An illustration of the increasing kind of phthisis-risk may be gathered from two diagrams that follow.

We have an article in our medical faith which says that chronic alcoholism causes an increased liability to phthisis. A trade which leads to the abuse of alcohol ought to give, therefore, a worse showing in the later years of life, when the alcohol has done its work, than even one of the worst of the non-alcoholic trades. The normal phthisis-risk is very bad in the case of the drapers. That of the publicans is still worse in the later years.

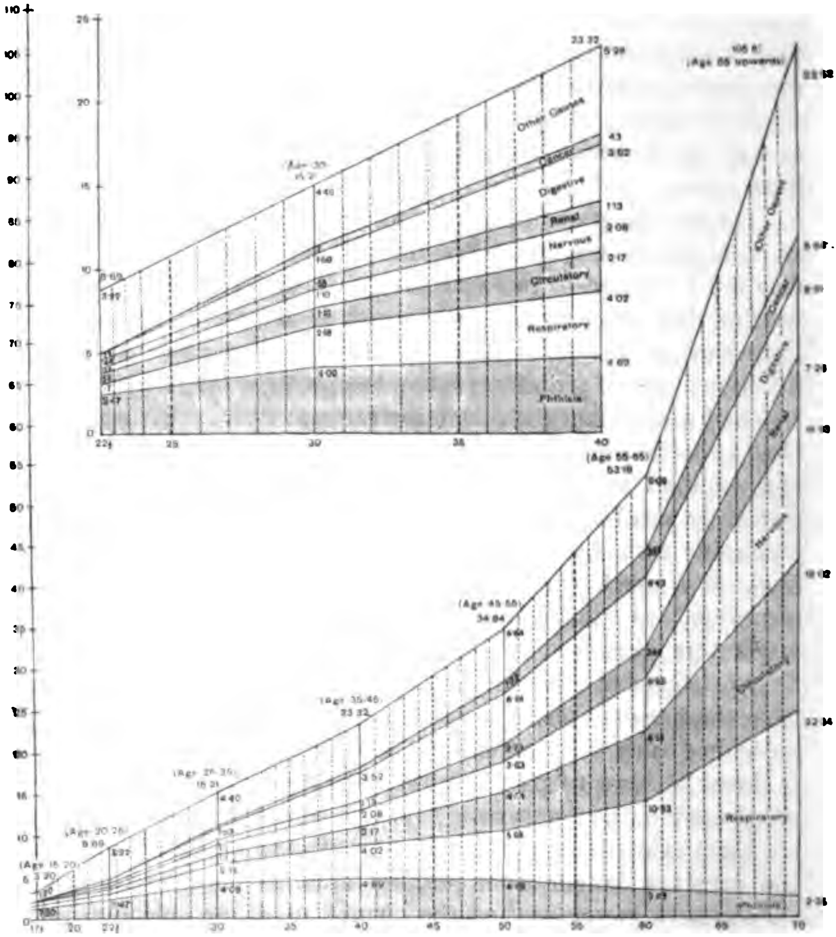
The two diagrams of these trades, which I have here, show an analysis of the mortality on the same lines as the first diagram I referred to this afternoon. If we compare the two, we find that the drapers' phthisis-risk is at first a little higher than that of the publicans, but after about the age of 45 the publicans' risk of phthisis is larger than that of the drapers, and this bad preëminence is maintained to the end. This suggests that a special influence is at work causing an undue increase in the acquired vulnerability of the publicans, and that cause is obviously alcohol. It must be noticed that these phthisis-risks increasing with age are often associated with other increasing risks of a much greater magnitude in making up the total mortality, as, for instance, the circulatory, nervous, and respiratory diseases.

An excessive constant danger from infection arises chiefly from the personal physique, as in the narrow and flat-chested individual with poor expansion and under weight, particularly if there be signs of past affection of either lung, or a history of pleurisy or hæmoptysis.

Such evidence causes the proposer to fall into a class where the constant danger from phthisis must be considered to be very greatly increased, and the chance of a new infection, or, what is the same thing, the recrudescence of an old one, to need a considerable addition to the premium, and sometimes rejection. Clinically we are unacquainted with immunity to phthisis resulting from an attack. As insurance examiners we are bound to consider that any attack, however slight, often precedes another, usually of a grave character. Hæmoptysis should always disqualify absolutely for several years, usually about 10. Not very long ago a case was sent in with carefully-

The Constitution of the Normal Risk in the Liquor Trade.
7,520 deaths.

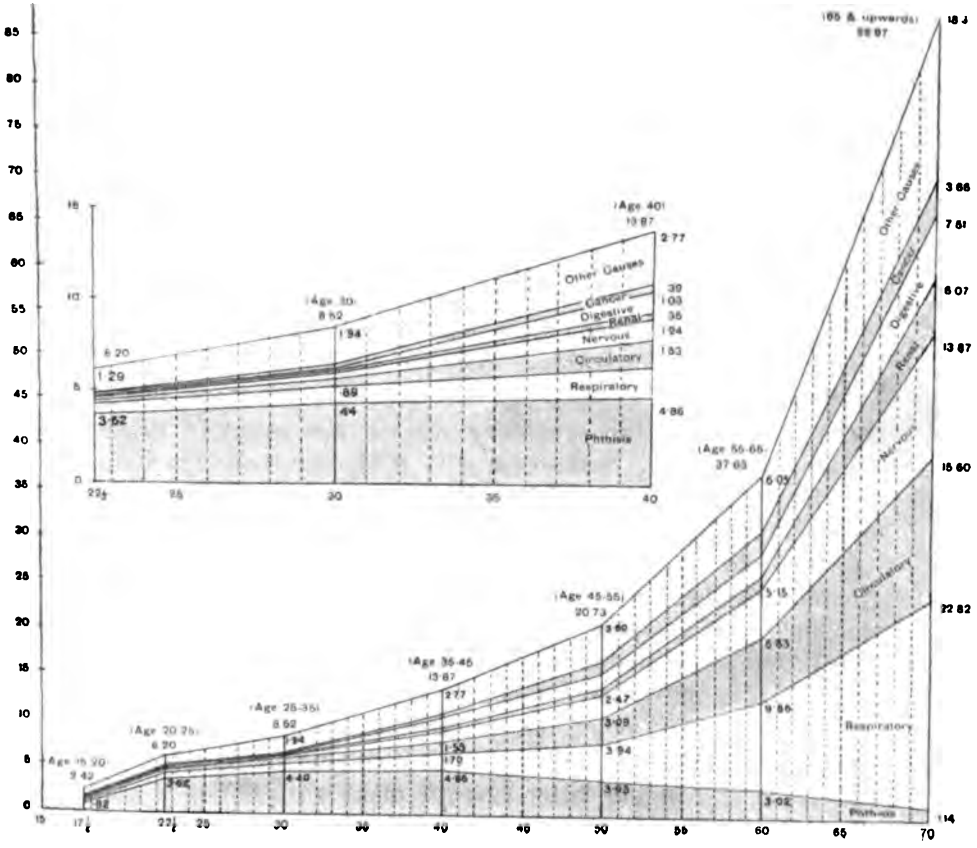
The risk at any age is built up of parts as shown.



described physical signs of arrested phthisis of recent date, and with a recommendation that the proposal be accepted at average rates because there were no signs of active change at the time. That recommendation was not followed at the central office, I need hardly say, for the fact of past mischief is a strong presumption that more may be expected, and it is impossible to regard a case for insurance in the same way as one would regard a patient. The one form of practice deals with future pathological tendencies, the other with present pathological states.

The Constitution of the Normal Risk in Drapers and Warehousemen.
2,100 deaths.

Note.—The vertical scale measures deaths per 1,000. The risk at any age is built up of parts as shown.



Finally, we come to the possibilities arising from there being an increased danger from both hereditary and acquired predisposition, or from one or other of these added to an increased danger from infection. You will have recognised that the attempt to classify the various risks has necessitated the separation of conditions which, as often as not, one finds combined in actual practice. I would say that hereditary predisposition with poor physique on the one hand, and acquired predisposition with a doubtful personal history of previous infection on the other hand, may be regarded as the two chief groups of combined dangers.

With such a broad grouping one could say that phthisis-risks may practically be regarded as of two kinds, decreasing and increasing, for obviously an addition of a constant to either does not affect its tendency to decrease or increase as age advances.

This classification is chiefly of importance in regard to the way in which the case should be dealt with at the office. Briefly, the addition recommended by the medical examiner is applied in different ways according to the tendency of the extra danger anticipated. If it be of a decreasing tendency, the addition can be perhaps best applied as a decrease in the value of the policy, of some considerable amount at first, gradually diminishing as age advances for a number of years, usually specified, after which the full value of the policy would be payable. As, for instance, a policy of 1,000*l.* might be reduced by 100*l.* in the event of death occurring in the first year, by 90*l.* in the event of death in the second year, and so on, till, if the proposer lived for ten years or more, the full policy of 1,000*l.* would be paid in the event of death.

It would sometimes be useful if the medical examiner were to indicate whether the extra danger from consumption which he finds in a proposer would, in his opinion, diminish with advancing age, actually or only relatively.

But it is not usually necessary for the medical examiner to do more than to point out whether the case is one to be regarded as an average life, or as one involving an extra risk to the office, and in the latter case to make an addition to the age. In forming this opinion the following points are, as I

A TABLE of the Factors to be considered in various Kinds of excessive Risk from Phthisis.

A.—HEREDITARY PREDISPOSITION.	B.—ACQUIRED PREDISPOSITION.
(1.) Requires a larger addition than family infection from which it should be differentiated.	(1.) Is to be inferred when there is a history of winter cough, bronchitis, recurrent pulmonary disorders of any kind, repeated attacks of influenza, or any depressing condition leading to malnutrition, chronic dyspepsia, or anæmia. The nature of the affection affects the rating.
(2.) Resemblance to the infected members of the family suggests hereditary predisposition.	(2.) Is increased by bad habits, especially as regards alcohol.
(3.) One or two cases in a large family less important than in a small family.	(3.) Arises from prolonged exposure to bad environment, as from such occupations as shopkeeping (especially drapers, hatters, tobacconists), hotel service, liquor-trade, commercial clerk, dusty trades, marine stewards and officers below deck.
(4.) Poor personal condition with a family history requires the largest addition.	(4.) Is diminished by robust physique.
(5.) Robust personal condition with a family history requires a smaller addition than when the personal condition is indifferent, and much smaller than (4).	(5.) Is increased by poor physique.
(6) A hereditary predisposition to phthisis late in life is occasionally seen.	(6.) Endowment assurance may afford some protection to the office by cutting off the risk in the late years.
(7.) Endowment assurance affords no protection to the office, as the extra mortality inferred in this class of proposer would come within the endowment period.	
(8.) Good environment diminishes the risk (especially when the personal condition is robust), *e.g.*, agriculturist, marine officers on deck.	
(9.) Removal from family infection should be ascertained before acceptance.	

C.—THE CONSTANT DANGER OF INFECTION.

(1.) Is increased by co-dwelling with consumptives. In view of the modern knowledge it is curious that this risk is not enquired into by the offices.

(2.) Is increased by poor physique, such as underweight, decreasing weight, poor development and expansion of chest, flattening of the chest, localised lack of mobility and asymmetry. Rapid pulse, existing cough or catarrh, debility, anæmia, dyspepsia with constipation, with poor physique often require postponement.

(3.) Is greatly increased by a history of past tubercular or possibly-tubercular pulmonary disease, and such a history always demands a large extra.

Any existing cough or catarrh demands postponement, especially with localised physical signs. Hæmoptysis disqualifies for several years. An arrested attack (which can often only be inferred from the unsatisfactory account given of a past illness) disqualifies for several years.

Old glandular or bone-disease in childhood that has never recurred does not markedly increase the risk, especially if the personal conditions and environment are good.

4. Congenital heart-disease or syphilis predispose to tubercle.

5. Apparently robust health is not incompatible with the presence of physical signs of active phthisis requiring disqualification.

have tried to illustrate by a method of first principles, the ones to be taken into account.

The normal danger to the office arising from phthisis is made up of three factors :—

Inherited vulnerability, largely diminished after middle life, *i.e.*, decreasing with age.
Acquired vulnerability, increasing with age.
Infection, equally likely at all ages.

The sum of these three factors causes the normal danger from phthisis to be the same at all ages, but as age advances the percentage of deaths from phthisis out of the total number of deaths diminishes with growing rapidity, owing to the larger number of other causes of death which occur in the later years. All additions for any extra risk from phthisis must therefore be larger in early life, but especially so with the danger decreasing with age. An excessive danger from phthisis may be inferred in an individual when he has inherited vulnerability, acquired vulnerability, or a risk of infection (or two or all of these) in a degree greater than average healthy members of his own class. Personal condition is an important factor in determining this degree.

There are several points to be considered in relation to each of the three factors, and for the sake of brevity I have put them in a table. They may be of occasional assistance in making a recommendation as to the nature of the extra risk among what are sometimes very difficult under-average lives to assess fairly.

In conclusion, I would borrow a phrase that has been used of men in general by their rulers, and would say that it is very true of insurance proposers in particular, and that phrase is that "every man has his price."

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DISEASES OF THE RESPIRATORY TRACT.

A REVIEW OF RECENT WORK.

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PULMONARY SYPHILIS.

WHILE lesions of the lung in congenital syphilis are not so very infrequent, pure cases of acquired syphilis of the lung are certainly very rare, and many examples which once passed muster have been discovered since the detection of the tubercle-bacillus to be in reality tubercular. Being so rare an undoubted instance, such as that reported by Winfield,[1] is interesting and valuable in recalling its existence.

Acquired syphilis of the bronchi is fairly common in the secondary stage as a bronchitis, in the tertiary stage in the shape of gummatous ulceration leading to stenosis. Syphilitic disease, though rare as a pulmonary lesion, does occur; and it may be that its known rarity leads to its being overlooked, and instances being confounded with tuberculosis. The results of the two diseases on the lung are in many respects so similar that no doubt of their tubercular nature ever arises, and an opportunity for striking improvement by treatment is lost.

Only a routine examination of the sputa in all cases will enable us to detect the non-tubercular processes. Consolidation of the lung occurs in syphilis by interstitial fibrosis involving the alveolar walls, associated with fibrinous pneumonia of the alveoli themselves. Gummata are also found, and both gummata and consolidation tend to caseate and break down, forming cavities which only differ from the cavities of tuberculosis in their tendency to affect the lower lobes and the parts about the root of the lung.

The case reported by Winfield is that of a woman, aged 38, who consulted him for gummatous ulceration of the skin. On enquiry a history was elicited of a miscarriage five years before followed by a sore throat and falling out of the hair. About a year after the miscarriage, cough and dyspnœa started, and she lost weight rapidly, falling from 135 to 95 pounds. Tuberculosis was diagnosed, though the sputum was never examined ; and she was advised to remove to the mountains. There she was treated for tuberculosis, until, falling into the hands of a skin-specialist, the nature of the skin-lesion was recognised, and the sputa were examined and found free from the tubercle-bacillus. The physical signs were those of a cavity at the right base. The absence of the bacilli was confirmed by three bacteriologists.

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[1] Pulmonary Syphilis, Report of a Case, *Medical News* August 30, 1902.

On specific treatment she gained weight, and became practically well, only slight cough remaining, with some dulness over the site of the cavity.

Berg's case[1] falls into a different category and raises a different question. The patient was a man, aged 28, who had had a chancre eight years before. For two years he had suffered from cough with thick expectoration, fever, and hæmoptyses, and had lost 30 lbs. in weight. During that time he had been under treatment, presumably directed to the cure of tuberculosis, but without effect.

The physical signs were those of consolidation at the right apex, reaching to the third rib, with a few râles. No tubercle-bacilli were present in the sputa. The treatment adopted consisted in a liberal quantity of milk, together with potassium iodide and perchloride of mercury. The man gradually improved and gained 20 lbs. in weight : the signs of consolidation passed away, only an occasional râle being heard, with slightly impaired resonance.

The signs being apical, one would have felt more confident about the case had the sputa been tested repeatedly. No mention is made of this. Still, as no tubercle-bacilli were found, and as improvement coincided with specific treatment, perhaps the diagnosis may be accepted. Gummata, though more common at the base, do occur at the apex of the lung, and such a case is mentioned by Dr. Fowler in his book.[2] In that case also tuberculosis had had been diagnosed. Tubercle-bacilli were absent from the sputa, however, and cure followed specific treatment.

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[1] Pulmonary Syphilis simulating Pulmonary Tuberculosis. *Medical Review*, ecember 13, 1902.

[2] Fowler and Godlee, *The Diseases of the Lungs*, p. 451.

Dr. Berg is a strong believer in syphilis as the determining factor in the development of tuberculosis of the lung, holding that previous syphilitic changes, such as endarteritis, are present, which form a nidus for the tubercle-bacillus. For him many cases of apical tuberculosis are syphilitic in the sense that the tubercle is implanted on a syphilitic soil. The connection between syphilis and tubercle and the frequency of tuberculosis in the syphilitic are undoubted, but it is more usual to refer the connection to the constitutional drain of syphilis lowering the resistance to tubercular invasion.

In the stress which Berg lays on specific treatment in these cases all would agree: as he says, nothing can promote the cure of the tubercle more than the removal of the deteriorating influence of the syphilis.

ETIOLOGY OF SEROFIBRINOUS PLEURISY WITH REFERENCE TO CYTOLOGICAL DIAGNOSIS.[1]

This subject has been dealt with in a previous review, but the matter is so important that fresh investigations and criticism must be welcomed and considered.

The importance of the subject dates from the discovery by the French that very many cases of simple so-called "idiopathic" pleurisy in healthy people, and even starting in a chill, are really tubercular in nature and may be followed by phthisis.

The French take up the extreme position (Landouzy, quoted by Bunting) that 98 per cent. of such pleurisies are tubercular, a position which, as he says, the Anglo-Saxon mind is slow to accept, still clinging to belief in the frequency of a simple exudative pleurisy non-tubercular and indeed non-bacillary. Without entering into a complete discussion of the whole subject, it will be sufficient to mention that, without accepting the extreme French position, investigation has conclusively shown that many at any rate of these cases are tubercular.

Kelsch and Vaillard (quoted by Bunting) found in 16 autopsies on cases of acute pleurisy tubercle of the pleura in all 16 (seven serofibrinous, two hæmorrhagic, four serous turning to purulent, three purulent from the first). Barr reports three similar cases in which tubercles were found on the pleura, death occurring a few months after the onset of an acute pleurisy.

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[1] C. H. Bunting, *Johns Hopkins Hospital Bulletin*, July 1903.

Such opportunities are, however, rare: though tubercular, such cases are not immediately fatal. Indeed, the immediate prognosis is very good, and recovery complete. The contention, however, is that a more insidious death is awaiting them, and that many of them will fall victims to phthisis or some other form of tuberculosis before many years are over. The percentage of these cases, which develop phthisis later, has again been the subject of much dispute, the figures varying according to the class of life of the patient, whether seen in hospital or private practice; but even the most favourable reports show a well-marked association between the two diseases.

Granting that some of these effusions are tubercular, and that the sufferers are threatened with phthisis, the problem is how to separate these cases from the non-tubercular pleurisies.

Bacteriological examination of the effusion lends but little aid, these serous effusions being usually sterile, in strong contrast with the purulent; or if microörganisms are visible on a coverslip, they are not cultivable owing to the bactericidal action of the fluid.

The tubercle-bacillus is scarcely ever found on coverslips, Bunting says, in these serous effusions. There is unanimous agreement on this point among all investigators.

Is the process then a sterile one, confirming the older view, or are the bacilli so few that search for them is like looking for a needle in a haystack? Animal inoculation has settled the point, for where considerable quantities of these apparently sterile effusions are injected into susceptible animals, tuberculosis follows in a large proportion of cases, varying from 50 per cent. upwards.

This method being inapplicable clinically, Widal and Ravaut have sought a basis for diagnosis in the nature of the cells in the exudate. They found themselves able to differentiate three groups according to the cell-contents:—

(1.) White cells mainly polymorphonuclear: but few uninuclear cells present and but few endothelial scales. The organisms present were streptococci, pneumococci, or the typhoid-bacillus ("acute septic" group of Widal and Ravaut).

(2.) In another group the white cells were mainly or exclusively uninuclear: few polymorphonuclear leucocytes and few endothelial cells, but many red corpuscles were present.

This group was found to be characteristic of the simple or idiopathic pleurisies, *i.e.*, the class among which the tubercular cases are to be found.

(3.) Mechanical effusions (hydrothorax) cardiac or renal. The distinguishing cells of this group were endothelial scales from the pleura, generally arranged in clumps (*placards*).

From their investigations Widal and Ravaut conclude that a uninuclear cell-content means tuberculosis. Wolff is in practical agreement with them, except that he would accept a lower uninuclear count (anything above 50 per cent.) as a test of tuberculosis. He pointed out that very early in this group the polymorphonuclear cells predominate: by the third day the uninuclear have increased to one-third of the whole, and ultimately become exclusive. The proportion of uninuclear cells then will depend on the age of the effusion, and one may note that Widal's 56 cases were all studied after the ninth day.

Bunting has studied 23 consecutive cases of serous pleural effusions in Osler's wards, not with a view of settling how far such effusions are tubercular—that, as he says, can only be determined by a large series of animal inoculations—but to see how far Widal and Ravaut's division according to cell-content holds good. In general his conclusions are confirmatory of theirs. In the group of primary idiopathic pleurisies he finds there is a very definite formula for the cellular exudate, the time at which it is taken forming, however, a marked factor in determining the proportions: in this he agrees with Wolff.

I. *Primary Pleurisies.*—(*a.*) At the end of the first week (3 cases) polymorphonuclear leucocytes 12 per cent. to 15 per cent.: rest almost entirely small uninuclear.

(*b.*) Second week (4 cases) polymorphonuclear cells 5 per cent. to 3 per cent. only.

(*c.*) After second week (6 cases) almost entirely uninuclear. Red corpuscles always abundant: endothelial cells scattered.

Two exceptions only were encountered :—

(1.) One was a terminal acute pleurisy in a cardiac case : the effusion was examined on the seventh day and was very hæmorrhagic and showed equal numbers of uni- and polymorphonuclear leucocytes. According to Wolff this might be tubercular, but it also suits the findings in an infarct (Barjon and Cade), and to this view Bunting leans. No autopsy was allowed.

(2.) A man was attacked one week before admission with a chill, fever, pain in the side, and cough with bloody sputa. On admission there was no fever and the signs were those of an effusion only. A cell-count showed 45 polymorphonuclear leucocytes to 55 uninuclear, along with abundant epithelial scales. He concludes that the case was one of slight pulmonary involvement. In none of these 15 cases were tubercle-bacilli found in the exudate : cultures were taken in many instances, but all were negative.

In two cases of pyopneumothorax secondary to tuberculosis of the lung tubercle-bacilli were found in both : polymorphonuclear leucocytes formed over 80 per cent. of the cells present.

II. *Septic Group.*—One case of metapneumonic purulent effusion. The pneumococcus was present in the exudate : polymorphonuclear cells constituted 96 per cent. ; uninuclear, 4 per cent.

III. *Mechanical.*—(Three cases, mitral regurgitation, chronic cardiac failure, and chronic nephritis.) Death occurred in all ; no tubercle was found *post mortem.* The exudate showed a large percentage of endothelium ("placards" abundant), but it is noteworthy that uninuclear leucocytes formed 96 per cent. to 99 per cent. of the whole number of cells present. Evidently, then, it may be very difficult to tell a hydrothorax from an idiopathic pleurisy with effusion, except by the surroundings. Fortunately, as the author says, the diagnosis would not often be in doubt. The point, however, is one that requires extended investigation, as it bears very strongly on the value of the method.

The author would have greatly added to the value of his work had he made inoculations from the effusions.

PULSATING EMPYEMA.[1]

Two cases of pulsating empyema recently reported are of special interest—Bendix' case, because the empyema was right-sided, whereas in an overwhelming proportion of cases it has been left-sided, only three cases of right pulsating empyema having been previously recorded; Henry's, because there were three pulsating tumours. In this feature it was almost unique, only one similar case being known.

The condition may be one to cause anxiety from the suggestion of thoracic aneurysm raised by the pulsation. Sometimes an actual tumour or pulsating protrusion of the chest-wall is present; in other cases there is no protrusion, but pulsation of the chest-wall can be felt, and seen.

In Bendix' case the patient was a woman who one month before had been seized with pleurisy. The right side was motionless on respiration, and dull from the middle of the scapula behind and the third rib in front downwards. Pulsation could be felt most prominently in the posterior axillary line, reaching from these nearly to the sternum over the space between the third and seventh ribs. It could be felt more feebly behind, and was synchronous with the heart-beat. The intercostal spaces were obliterated over the pulsating area. The breath-sounds were absent at the base, and the heart was displaced nearly to the left anterior axillary line. Its impulse was leaving and there was an apical systolic murmur. In short there were the signs of a large effusion which pulsated: the heart was displaced in the usual way, but was itself diseased and hypertrophied. The Röntgen rays showed a dark shadow, reaching to the third rib, continuous with the heart's shadow on the left: this dark shadow pulsated strongly below, but its upper edge was motionless.

Exploration in the pulsating area brought away very thick pus. On draining the empyema 300 cc. of thick pus were evacuated, and probing showed the lung to be lying far back and collapsed. The next day thick purulent masses were discharged in abundance.

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[1] "Pleuritis pulsans dextra." Bendix, *Münch. Med. Woch.*, May 19, 1903 "Pulsating Empyema Necessitatis." F. P. Henry, *New York Med. Journ.*, May 9, 1903.

Bendix thinks the necessary conditions for producing pulsation are a powerful action of the heart, which in his case was hypertrophied, and some means of transmitting the pulsation to the chest-wall; but this need not in all cases be the same. In his case a firmly-encapsuled effusion, which itself was unusually thick in consistency, produced a condition almost amounting to a solid tumour. In other cases a collapsed lung adherent to the chest-wall and in contact also with the pericardium gives the necessary condition. Many of these cases are cases of pyo-pneumothorax. Féréol suggested that the air was strongly compressed and formed an elastic cushion which transmitted pulsation.

In two of Traube's cases the costal pleura was eroded; this is not invariably the case by any means, but it has been supposed that some relaxation of the thoracic wall from paresis of the intercostal muscles may occur and take part in the production of the pulsation.

The *diagnosis* from aneurysm Bendix does not consider difficult, the absence of a systolic murmur over the pulsating area being an important point. The signs also are often those of a large effusion or a pneumothorax. The Röntgen rays may be very useful, especially if, as in Bendix' case, it is found the shadow does not pulsate in all directions.

In Henry's case two of the expansile tumours were dull, but one was resonant, and a splash was evident in it on palpation, *i.e.*, it contained air as well as pus. There were signs of a general pyo-pneumothorax, and the pus, as in Bendix' case, was noted to be extremely thick—so thick that it could hardly be poured out of a vessel containing it.

ALBUMINOUS EXPECTORATION FOLLOWING THORACOCENTESIS.[1]

The title, though describing the most striking features of the condition, gives but little idea of its gravity. It is always a sign of great danger, and in one of these cases death resulted. The condition is really one of acute œdema of lung, the result of its release from the collapse in which it has been held by the pleural effusion. The albuminous expectoration is the transudation from the vessels of the lung, which wells up in such abundance that the patient is in danger of asphyxia.

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[1] A. J. Patek, *American Medicine*, August 23, 1902. W. C. Drein, *American Medicine*, January 3, 1903.

The first symptom of the occurrence is cough with the expectoration of a thin, frothy, straw-coloured fluid, which soon becomes profuse. In one of these cases 2 ounces were coughed up in the first twenty minutes, and later the fluid is described as gushing in a steady stream from the mouth and nostrils; in the other case the amount expectorated was estimated at 750 cc. The cough becomes incessant; cyanosis and intense dyspnœa follow; the pulse is rapid and feeble, and all the signs of cardiac failure are present. On auscultation innumerable small and medium-sized râles, characteristic of œdema, are heard over the affected lung. Occasionally these symptoms occur during aspiration, more often a short time—half an hour—after. Both the reported cases were unusual in their being delayed to two hours after.

The expectorated fluid is highly albuminous. In this and in its colour it bears a strong resemblance, which all writers have noted, to the aspirated pleural effusion. On this similarity was based the theory that the fluid expectorated was the pleural effusion, which had broken through and flooded the lung. The two, however, on analysis, have been found not to be absolutely identical in composition, and no trace of rupture has ever been found *post-mortem.*

The explanation given by the authors is the one usually accepted, viz., pulmonary œdema from acute congestion (Riesman's "Congestion by Recoil"), induced by the rapid expansion of the lung. The accident has nearly always occurred in cases of large effusion, and the two quoted cases are no exception to the rule. In many of the cases, too, the amount of fluid withdrawn has been large, and it is obvious that a large withdrawal, especially if rapid, must favour the occurrence of pulmonary œdema. In one of these cases, however, no great quantity (1,620 cc.) was withdrawn; in the other, 3 litres.

Stimulants and wet cupping are the best treatment, though in one of these cases they proved unavailing.

THE SHAPE OF THE PHTHISICAL CHEST.

Under the title "Is the Phthisical Chest flat?" Woods Hutchinson[1] restates his views, and adds further observations in support of his position.

His view is entirely contrary to received opinion; to quote his words, "the tuberculous chest instead of being flat is really round, *i.e.*, the antero-posterior diameter instead of being diminished relatively to the transverse is, on the contrary, increased, so that the term flat is a misnomer." All the text-books and authorities, except one, adhere to the received view, a mere parrot-like repetition, he thinks, on their part.

Method.—To obtain the relation between the two diameters of the chest he measures them by callipers placed, for the antero-posterior diameter, with one point on the sternum at the level of the nipple, and the other on the dorsal spinous process horizontally opposite; to obtain the transverse diameter the callipers are slid back at the nipple-level till the widest possible point of separation is reached: both measurements must be taken in the post-expiratory pause or resting position.

The transverse diameter is taken as 100, and the antero-posterior is expressed in terms of this, giving what he calls his "index." From measurements of about 50 normal people he finds that the antero-posterior diameter or depth is about 70 per cent. of the transverse or width, *i.e.*, the normal index is 70. Within this average figure there are some exceptions; a few will have as low an index as 62 or 63, a few as high as 80.

The index varies at different ages. At birth the diameters are almost equal (index 101), *i.e.*, the normal infantile chest is round; with increasing growth and exercise he has traced the chest gradually becoming broader.

Under 2 years	the index is	94.
Between 3 and 7 years	,,	85.
Boys from 14 to 18 years	,,	80.

In women there is normally a greater persistence of the infantile type than in men.

Applying this method to the tubercular chest he was surprised to find that the average index was higher than the normal, *i.e.*, that, contrary to the usual belief, the phthisical chest was not flat, but rounder or deeper than normal. In 40 tubercular cases examined in America the average index was 79½; in 42 cases examined by him in the wards at Brompton it was 79·6.

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[1] *Journ. Amer. Med. Assoc.*, May 2, 1903.

This result has been confirmed by various independent observers, and their figures are given in this paper.

Colbeck, at the Victoria Park Hospital (London), found the average index of 75 cases to be 80·3—males (61), 79·3; females (14), 84·3. Of the 75, the disease was in an early stage in 42; their index was 77·6. From this he deduces the interesting explanation that the tubercular chest is a persistent immature chest, preserving the infantile type: to this, no doubt, the proneness to the disease is due, and it very likely corresponds to an immature type of constitution.

From Colbeck's early cases he infers that this type of chest precedes and leads up to the disease, and is not produced by it. Phthisis is not, however, confined to this type of chest, though the majority of high-index cases is overwhelming. In another series of 35 tubercular cases eight had an index of 70 or below, four reaching as low as 63 or 64. One would think that these represent the group of cases who, without constitutional predisposition, fall victims through ill-health, privation, or repeated opportunity of infection.

HYDATID OF THE LUNG.

Hydatid disease of the lungs has lately been made the subject of an excellent monograph[1] by A. A. Lendon, of Adelaide. Abstracts of a well-selected series of cases illustrating all phases of the disease, including its chief complications, are incorporated, and in addition there are chapters on Hydatid of the Pleura and the Invasion of the Thorax by Abdominal Cysts. The book is evidently written from a large clinical experience, and may be taken as a trustworthy guide.

It has been usual hitherto to divide the course of the disease into two stages, and two only—the stage of the unruptured cyst and the stage after rupture, the latter being marked by all the signs of destructive lung-disease, fever, profuse and often fœtid expectoration, and wasting. Lendon, however, adopts a more elaborate classification as follows :—

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[1] *Clinical Lectures on Hydatid Disease of the Lung.* By A. A. Lendon, M.D. Lond. Baillière, Tindall, and Cox, 1902.

a. *The unruptured Cyst.*

Stage i. Latency.
Stage ii. Suspicion (marked physical signs).

b. *The Rupture of the Cyst.*

Stage iii. Rupture.

c. *The ruptured Cyst.*

Stage iv. Quiescence.
Stage v. Expectoration of Mother-Cyst (patient may die of impaction of the membrane in the glottis or of hæmorrhage, or may recover with a potential cavity).
Stage vi. Sepsis.

d. *The empty Sac.*

Stage vii. Empty Lung-Cavity.

Unnecessarily elaborate as this division may be, his treatment of the Ruptured Cyst (*c*) and his division of it into three stages is original and full of merit. The symptoms mentioned above, usually described as associated with the ruptured cyst (fever, wasting, &c.), are really septic and are due to infection of the cavity by pyogenic and other organisms from without. Such a septic infection may set in at any time, early or late, after the rupture, but it need not necessarily occur. Lendon recognises this, and supports his belief by a striking case. Not only may the ruptured cyst remain quiescent and give no signs of its presence, but, what is more striking, it may break up and be expectorated without sepsis or fever, the general health even improving, until ultimately a natural cure may be reached. The question of sepsis or non-sepsis he considers to depend on the condition of the bronchi at the time of rupture, and the presence or absence of organisms in them which might invade and infect the cavity.

It is under this condition of non-sepsis that the many reported cases of cure by a single tapping have been effected.

A period of quiescence, which he defines as absence of all symptoms, though physical signs are present, is the usual condition after rupture, the cyst being collapsed and rolled up, and the lung having largely reëxpanded and closed the cavity as soon as the pressure was relieved. The longest period to which he has known quiescence to be extended is two and a half years, and he mentions another case in which it lasted a year.

After a time this aseptic cyst breaks down—as he thinks, mechanically from cough—and is expectorated. Cough, with fairly profuse, but not fœtid, expectoration, is now present, and sharp hæmoptyses are quite a feature of this stage. Fever, however, is absent, and the other signs of sepsis; and ultimately, as stated, the cough may cease and a cure be reached. Lendon does not advocate operation very strongly while this stage persists; there is not the same imperative need for it as in the septic stage, and the cavity having reëxpanded the physical signs are often obscure and the cyst hard to locate. The risk of asphyxia from blocking of the glottis by a piece of membrane, or of fatal hæmoptysis, is apparently about balanced by the difficulties and risk of operation.

In reviewing the other stages one may pass over Stage I. (the unsuspected cyst) except to note that a cyst may reach the capacity of a pint without attracting attention, but this is usually only because it is masked by disease elsewhere. In this stage of latency the hydatid may die, but this is excessively rare: usually it obtrudes itself on the attention either by rupturing—which may be the earliest and only symptom—or more commonly by its increasing size.

In Stage II. (the suspected cyst) cough, hæmoptysis (due to vascularity of the lung round the cyst), and dyspnœa are all frequent symptoms, there is no fever or malaise. The physical signs are bulging (only met with in children), absent vocal fremitus, and absent breath-sounds. The percussion note may be dull or tubular. It is interesting to note that hydatid fremitus, rare at any time, is rarest in thoracic cysts, and Lendon has only twice met with it; also that he lays no stress on a curved edge to the dull area in distinguishing a hydatid from a pleural effusion.

As to pressure-signs, he has never found the abdominal viscera displaced to any notable degree, and cardiac displacement is never very great, even with cysts which occupy the lower half of the chest, the reason being that the cyst is contained within the lung and does not compress it from without.

In this stage he holds that a certain diagnosis is impossible in the absence of bulging, but a high degree of probability can be reached. At the apex the differential diagnosis is from tuberculosis, at the base from pleural effusion. In the case of the latter it was mentioned that a hydatid causes less cardiac displacement, and in addition the breath-sounds are dissimilar, the tubular breathing at the upper level of fluid not being met with in the case of a cyst.

In operating at this stage—and tapping has been discarded because of its peculiar danger—he adopts certain precautions. To avoid the danger of flooding the lung, the anæsthetic is lessened and the patient allowed almost to come-to, just as the rib is removed, in order that cough may be preserved; with a similar end, as he incises the cyst, he turns the patient over, making the wound the most dependent part. He finds it better to drain the cyst in order to avoid emphysema and to evacuate the blood from the cavity. The cyst, as is well known, is easily removed entire, as its attachment to the lung is so slight. Lendon utters a caution with regard to pleural adhesion; he finds it often absent even in cases of old cysts abutting on the surface of the lung.

Under the head of "Rupture" is described the flooding of the lung with fluid, which is the great danger in the aspiration of a cyst. His explanation is that the needle splits or lacerates the cyst and the fluid escapes with a gush. Lendon himself has had an experience of this accident, and never aspirates now without having a knife ready, immediate incision of the cyst being the best and only treatment.

In the case of septic infection after rupture he operates in practically the same way as for the unruptured cyst; drainage, however, has to be maintained longer—indeed, until the discharge becomes serous or ceases. In the absence of adhesions he is more careful to drain the pleura.

The empty cavity in the lung after evacuation of the cyst sometimes fails to close and may need drainage; this is more frequent after spontaneous evacuation, when septic processes are more liable to occur.

THE TREATMENT OF NEPHRITIS: SOME RECENT SUGGESTIONS.

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THERE are few patients the treatment of whom taxes the physician's skill more than those suffering from nephritis. Yet there is probably no disease in which treatment, based upon a sound knowledge of the patient's condition and experience of the effects of therapeutics in other cases of the malady, has a greater influence upon the course of the disorder. There is a tendency to regard diseases in which drugs have little or no good influence as being to a large degree outside the sphere of active help on the part of the doctor. To those who yield to this tendency affections of the kidney, other than those amenable to surgical interference, are apt to sink into that class of diseases which is met by mere expectant treatment. This is especially the case with acute nephritis, because here drugs are notoriously of little avail. But when it is remembered that, next to disorders of the digestive tract, diseases of the kidney are more than all others directly influenced by dietetic considerations, and that these considerations are wholly in the hands of the physician, it follows that the scope of treatment in nephritis is in reality very large and important.

It may be readily granted that our very scanty knowledge of the processes of metabolism and nutrition puts a serious limit to the rational treatment of disease of the chief organ of elimination. But if we are willing to accept indications from every available source of knowledge—foremost, perhaps, from careful experience of actual cases of kidney-disease, especially when modified by the effects of treatment, but also from investigations into general tissue-metabolism—we shall find that the power of combating the various forms of nephritis grows considerably. This is, of course, equivalent to advancing a plea for fuller metabolic studies in the cases of nephritis under our care; and this plea we make with the conviction that it is only along these lines that any advance in therapeutic knowledge can be expected. The difficulty—the impossibility often—of carrying out any elaborate investigations of this kind in the case of private patients is admitted. The very attempt, often revealing an unusual interest in the case, has a powerful psychic effect on the patient, which, at first tolerated or even welcomed, soon becomes tedious and later quite undesirable from its power of suggestion. In hospital out-patients, again, the matter is quite impracticable. But with cases in hospital, where the routine of the wards lends every assistance to the work, and the patient quickly learns that the atmosphere is one of constant investigation, it is sad to reflect how few thorough observations are made upon the liberal supply of material at hand. With the laboratory close by, what more useful training could a research-clerk have than to study a case of acute nephritis for a period of days from the point of view of ingestion, metabolism, and elimination? Some of the matters needing investigation may readily be deduced from the remarks which follow.

The efficient study of cases of nephritis has lately received a great stimulus in the forn of the publication, first in America, and then in England, of a translation of Professor von Noorden's monograph on this section of *Disorders of Metabolism.*[1] After a preliminary sketch of the customary form of therapeutics in nephritis, the author discusses the principle of saving the kidneys in disease of these organs, and admits that it must be the basis of all successful treatment. But the reader is reminded that the points which guide the physician in estimating the degree to which any diet spares the kidney in nephritis are by no means simple.

The chief point is *the course of the disease as modified by different dietaries*, as we have already hinted. The *degree of albuminuria* must be interpreted with caution. Perhaps it is only in acute nephritis that the amount of albumen is a conclusive index of good or bad results; here, certainly, a rise or fall in the albumen usually accompanies an exacerbation or remission in the general condition. In subacute cases with œdema, periodic flunctations in the amount of albumen may occur quite independently of the diet. In the contracted

[1] Part II., *Nephritis.* Translated by Boardman Reed, M.D. New York: E. B. Treat & Co.; Bristol: John Wright & Co. 1903.

kidney the albuminuria is notoriously fallacious as a guide. Any sudden change in the diet is apt to cause an increase in the output of albumen; moreover, a high figure is often indicative of an acute exacerbation rather than related to the permanent degree of kidney-damage. The third criterion is *the excretory power of the diseased organ.* This may be tested in various ways. The method advocated by von Noorden is to study the elimination of the food-principles themselves.

ACUTE NEPHRITIS.

As the result of many observations in cases of acute nephritis the author classes the following amongst the substances excreted with difficulty: urea, creatinin, pigments, hippuric acid, phosphates, inorganic sulphates. The substances well excreted are: uric acid, xanthin-bases, aromatic substances, ammonia, amido-acids, chlorides and carbonates. As regards water, the important observation is made that in the early stages of acute nephritis water is eliminated with great difficulty; later on, this difficulty passes away. Coming to the practical issue, the dietary recommended in acute nephritis may be set out in the following scheme:

1.—*To be avoided:*
  (i) Because of creatinin produced:—meat-extracts, meat-broths, eggs.
  (ii) Because of pigments produced:—foods containing hæmoglobin.
  (iii) Because of hippuric acid produced: — green vegetables.

2.—*To be given sparingly:*
  (i) Because of urea and sulphates produced:—milk.
  (ii) Because of irritant effect during excretion:—water.

3.—*To be given with impunity*, in all but severe cases, because no injurious end-products are produced:—cream, butter; white vegetable soups; puddings made from oatmeal, flour or maize; white bread.

To neutralise the bad effects of the phosphates in milk, a little calcium carbonate is added; this enables the phosphates to be got rid of in the fæces.

It will be seen that the traditional practice of giving an exclusive milk-diet to all cases of acute nephritis is not supported by von Noorden; nor does he advise the unlimited supply of fluids usually recommended in all cases.

(*a.*) *For severe cases in which there is partial or complete suppression of urine, increasing œdema and threatened uræmia,* the author allows only a pint of milk a day, and avoids other fluids. Thirst is relieved by ice or very small quantities of water. A little alcohol is allowed. Free diaphoresis is encouraged.

(*b.*) *After four or five days,* or *in cases less severe than* (*a*), such as are common during or after acute infectious diseases, milk up to two pints and a half is allowed, with calcium phosphate added (*vide supra*), and half to three quarters of a pint of sweetened cream, previously sterilised. If more food is required, no more milk is given, but articles are added as in (3) above. Fluids are still restricted so long as there is oliguria and œdema, and diaphoresis is favoured; but if the excretion of water is good throughout, or has returned after being deficient, fluids should be given freely. If the œdema is passing off, diaphoresis is unnecessary.

(*c.*) *The convalescent stage* demands more food and a more varied dietary. White bread, *légumes*, cream-cheese, a little egg or meat may be added, taking care to keep the total amount of proteïd below 100 grammes *per diem*. At this stage fluid will be borne well and does much good. If œdema reappears, the skin must be stimulated or a dry climate chosen for the patient.

In all stages diuretic drugs, whether acting upon the heart or directly upon the kidney, are of no use.

THE CONTRACTED KIDNEY.

In the author's discussion of the treatment of contracted kidney, he points out that the condition is very different from acute nephritis. The kidney must again be spared as much as possible, but the whole organism must be fortified, and especially the heart-muscle must be spared and strengthened. Some articles of food must be entirely omitted: alcohol, condiments (except salt, vinegar and lemon-juice) such as pepper, ginger, mustard, spices, celery, radishes and asparagus. Certain drugs are to be avoided: salicylic acid, resorcin, lead, boric acid, mercury, iodoform and tar. Coffee, tea and tobacco are injurious in that they dissipate the energy of the heart instead of strengthening it. As regards meat, apart from excluding strong broths or extracts and "high" game &c., the author considers no further limitations should be imposed. The customary disinction made between "white" and "red" meats he holds to be erroneous, experience showing that the former is not a better article of diet for patients with granular kidney than the latter. His advocacy of a varied and fairly liberal diet for these patients agrees with the experience of many other physicians. But patients suffering from granular kidney are still not unfrequently condemned by their doctors for long periods to diets very poor in proteïds and very limited as regards a choice of articles of food; their general condition improves rapidly on a more varied dietary. Professor von Noorden considers that most cases of contracted kidney do better with a limited allowance of water. Flooding the tissues overtaxes the heart, and large quantities of water absorbed from the intestines can only be excreted by means of a high general blood-pressure. The author goes so far as to say that he thinks in many cases life may actually be saved by a sensible restriction of water. He places the total limit of ingested fluid at two and a quarter pints *per diem*. If this restriction of fluid is found to diminish the elimination of solids in the urine—this does not occur as a rule—one "drinking day" may be allowed in the week, to remove accumulated excretory material from the body. Even those patients whose atrophic nephritis was complicated by gout, according to von Noorden's experience, fared better with the limited allowances of fluids. As regards physical treatment, this should be carried out on the same principles as apply to cases of heart-disease.

The present writer has dealt thus fully with Professor von Noorden's monograph because he believes that the lines of treatment there laid down in cases of nephritis are the best at present known. They are receiving daily confirmation both from clinical experience and laboratory research. Kaufmann and Mohr (*Zeitschrift f. klin. Med.*, Bd. XLIV., p. 441) found that when cases of nephritis were put successively upon beef and veal, and the periods during which each was given changed abruptly, there was no appreciable difference in the nitrogen-output. They also found considerable advantage followed a due restriction of ingested fluid. Their experience of the indifference of "red" and "white" meats as forms of food for nephritic patients agrees with the results of Walker Hall's researches into "The Purin Bodies of Foodstuffs" (*Manchester: Sherratt and Hughes*, 1902), during which it was found that little difference exists between the amounts of purin-bodies in white and dark meats. The benefit derived from the addition of bread-and-butter, rice, fruit, or even meat, as soon as any acute process has subsided, is recorded by C. P. Emerson (*Johns Hopkins Hosp. Reps.*, 1902, Vol. X., p. 323) in an analysis of ten cases of nephritis. The author also draws attention to the fact that in governing the diet in this disease slight rises of temperature should be taken as evidence that the diet is unsuited to the case.

THE CURE BY DECHLORINATION.

In a communication made in June last to the *Société Médicale des Hôpitaux* by MM. Widal and Lemierre, these authors established a relation between the amount of chloride of sodium ingested and the œdema in two cases of tubular nephritis. The absorption of 10 grammes of salt *per diem* led to considerable œdema. In four cases of interstitial nephritis it was found that no such effect occurred. In a very striking article in *La Presse Médicale* (Juin 27, 1903) MM. Widal and Javal follow up this observation, and record the case of a patient suffering from parenchymatous nephritis whose ingestion and excretion of sodium chloride were estimated, and made to vary, over a period of seventy-two days. During this time nine abrupt changes were made in the chlorides of the diet; in five of these there was a balance of chlorides excreted, in four there was a balance retained. These events were brought about alternately. When the chlorides were retained, the weight increased, as also did the œdema and the albuminuria. When the chlorides were excreted, the weight diminished, as also did the œdema and the albuminuria. The authors conclude that salt is, for certain cases of tubular nephritis, a dangerous constituent of food, and they propose a "*cure by dechlorination*" for such cases. They ascribe the beneficial effect of a milk-diet, hitherto empirically employed, to its poorness in chlorides, and suggest the addition of several other articles of diet in the treatment of nephritis, provided no salt is used in their preparation. Indeed, the milk might be improved upon by choosing a still more largely "dechlorinated" diet. In the particular patient under observation, for instance, the authors found that a daily diet consisting of bread (without salt), raw meat, sugar, butter, and weak tea, containing only 1·5 grammes of sodium chloride, and continued for eighteen days, led to a total disappearance of the œdema present when this diet was commenced, and to a diminution of the albumen from 5·32 to ·75 grammes *per diem*. Without entering here into the very important bearing which such observations as these have upon the pathology of œdema, and the confirmation they give to certain recent views on this subject, it is obviously a matter for careful investigation, and suggests a line of treatment the success of which can only be judged after more extended experience.

FUNCTIONAL ALBUMINURIA.

Under the title "Curable Albuminurias," G. Lyon (*La Presse Médicale*, 5 Sept. 1903) discusses the treatment of cases of albuminuria more often termed "functional." Insisting upon the view that these patients are suffering from a constitutional and not a local disease—the inheritance bequeathed to them by parents who suffered from gout, gravel, diabetes, obesity, or megrim; in a word, ancestors whom the French term "*arthritiques*"—the author emphasises the importance of a *régime* that will alter the constitutional condition. This is effected by attention to the general health, but there is nothing in the detailed directions given as regards diet, exercise, habits, &c., which calls for special remark. In view of the marked effect of posture on the albumen in these cases the author advises rest in the horizontal position for an hour after meals. This is certainly an important point in the treatment, and an injunction against any prolonged assumption of the erect posture might have been added with advantage. Linossier and Lemoine have recently shown (*La Semaine Méd.* 1903, p. 113) that even in health the kidney-functions are interfered with by the erect position. If the kidneys are diseased, there is a diminution of 36 per cent. of the water excreted, and often a decrease in the urea also,

URÆMIA.

In the therapeutic management of the uræmic state, that most serious of all the manifestations of kidney-disease, the *value of venesection* is attested by several observers. Walko reports 11 cases of uræmia occurring in V. Jaksch's clinic treated in this way (*Zeitschrift für Heilk.*, Hft. 8 u. 9). It was found that only in acute nephritis or in the acute exacerbations of chronic nephritis were good results obtained, with the production of marked diaphoresis and of diuresis with decreased albuminuria. In chronic cases the method was quite ineffectual or only fraught with temporary relief. This is the experience of most physicians. Marsden (*Med. Chronicle*, October 1902) concludes that venesection in nephritis is specially indicated in those cases in which dilatation of the right heart follows overstrain and failure of the left ventricle, and records two cases where the good effect of the measure certainly seemed related to the mechanical relief of an overburdened circulation.

Diaphoresis only seems to be of use if it is followed fairly quickly by diuresis. As Walko (*ibid.*) points out, persistence in the production of sweating, without diuresis, may even be injurious; for examination of the sweat shows that it contains quite an insignificant quantity of nitrogenous substances. The blood-serum is thus in danger of being left in a state of higher molecular concentration. Heinrich Stern suggests (*Amer. Med.*, May 2, 1903) that, if sweating is kept up by artificial means, infusions of weak saline solution should be given either *per rectum* or hypodermically.

The same author reminds us that when *subcutaneous or intravenous saline infusions* are given, with or without venesection or diaphoresis, a hypotonic solution should be used, so as not to increase the already high osmotic tension of the serum. Therefore, instead of the ordinary isotonic solution (·9 per cent. NaCl.) used after hæmorrhage, &c., a solution not stronger than ·5 per cent. should be employed.

THE CLAIMS OF SURGERY.

Eighteen months ago the question of the operative treatment of nephritis was fully discussed in these columns. It was pointed out that so long as the term "nephritis" was loosely used to cover various local and unilateral kidney-diseases, as well as cases of Bright's disease proper, no satisfactory opinion could ever be arrived at as to the scope of surgery in the treatment of true nephritis. Since that time several surgeons have published records of operations upon genuine cases of Bright's disease, and the literature of the subject has grown considerably. There seems no doubt that in many cases improvement has followed the operations, but as yet it is too soon to say whether this improvement is permanent or not. Several of Edebohls' original cases are constantly being quoted as instances of cure after the lapse of some years, but it must be remembered that this surgeon particularly came under reproof for the meagre evidence adduced to show that these cases were anything more than instances of renal disease carrying well-recognised surgical indications. Edebohls still adheres to the notion that Bright's disease may be unilateral. In his last communication, "Renal Decapsulation for Chronic Bright's Disease" (*Med. Record*, March 28, 1903), referring to nine cases of unilateral chronic nephritis detailed a year ago, the author says:—"While surgeons of greater experience in renal surgery were, as a rule, ready to accept my findings as correct, in view of operative experience of their own pointing in the same direction, the contention was made by some clinicians of the highest rank that chronic Bright's disease is always bilateral. It appears to the writer that these two different positions and views are not so difficult of reconciliation as may appear on the surface. Chronic Bright's disease, *as such and in itself*, rarely or never causes death when limited to one kidney; in fact its unilateral existence, save in very exceptional instances, goes practically undetected during life, unless accidentally revealed either by urinary examination or by the knife of the surgeon. Surgeons, in their operative work, have now and then met the disease in its earlier stages unilaterally; physicians derive their convictions from the fact that the patient comes under their professional care in the later stages, when the disease has become bilateral, and from the observation that the disease, as found in the dead-house, is practically always bilateral. Bright's disease, like everything else, must have a beginning; that it may make decided progress in one kidney before attacking the other, I, in common with other surgeons, consider proven." But if these things be so, the question naturally arises: How is it ascertained which is the affected kidney? We are not told. If it be by the presence of pain or other local symptoms or sign, does not this suggest that the supposed unilateral Bright's disease is really one of the forms of kidney-mischief referred to above as having long been successfully treated surgically?

Edebohls no longer confines his operations to cases of chronic Bright's disease; he also treats cases of acute nephritis surgically, and Cavaillon and Trillat of Lyons have recently reported a case of puerperal eclampsia where Edebohls performed double decapsulation of the kidneys with a good recovery.

The question is dealt with by Le Nouëne (*Thèse de Paris*, 1903), and still more fully discussed by MM. Patel and Cavaillon (*Annal. des Mal. des Org. Gen-Urin.*, 15 Sept. 1903), who give a full bibliography of the subject. These authors give the following indications for operation in "nephritis":—(i.) Nephropexy in the albuminuria of displaced kidneys. (ii.) Puncture or incision of the capsule, in acute nephritis with great congestion. (iii.) Capsulectomy in chronic nephritis. (iv.) Nephrectomy, when one kidney is quite useless or when it is desired to prevent the reflex inhibition on a sound kidney by its diseased fellow.

Here, again, we find the old confusion arising. For indications (i.) and (iv.)—passing over the questionable point of the sympathetic effect of one kidney upon the other—refer to conditions by no means new in the realm of surgery. Still, the authors are convinced that the indications (ii.) and (iii.) are well supported by growing experience. They conclude that a new domain has been opened to renal surgery, but, unlike Edebohls, they are content to restrict the sphere of operation to such cases of Bright's disease as fail to be cured by the ordinary methods of treatment.

Some experimental work has been done with a view to elucidate the question of how the good effects claimed for such operations are produced. Johnson (*Annals of Surgery*, vol. 37, p. 592, 1903) performed the operation of renal decapsulation upon fifteen dogs and examined them at intervals of two days up to three and a half months afterwards. He found that in two months a new capsule formed, composed of homogeneous fibrous tissue, thicker than the original capsule. This is what might have been expected, and it will be interesting to see what will happen eventually to patients in whom this condition is produced as the result of operation. In no case did the author find any considerable anastomosis between the renal and perirenal blood-channels. These experiments, therefore, support the view of Israel and of Pousson, that the alleviation of the symptoms after operation is due to the relief of tension, not, as Edebohls thinks, to the production of a collateral blood-supply. It can be readily understood how the relief of tension in nephritis—at least in acute nephritis—would be beneficial; but whether so serious a mode of producing it as is here suggested is justifiable, remains a question. Unfortunately the situation of the kidneys precludes puncture being performed through the skin without too great a risk of perforating other viscera. If it were not so, this would be quite a sound therapeutic measure in some cases. And to puncture the kidney directly practically amounts to a major operation.

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TWO OBSTETRIC CASES WITH COMMENTS.

BY W. E. FOTHERGILL, M.A., B.Sc., M.D., *Manchester.*

THE two cases now reported both occurred in the practice of Dr. Carter, of Manchester, and are of interest amongst the curiosities of obstetrics.

I.

ACUTE HYDRAMNIOS WITH QUADRUPLETS.

Case I.—Mrs. X. is a healthy woman, 37 years of age. Her family history is of no interest in the present connection, as it affords no examples of plural conception; nor does her husband remember the birth of twins or triplets to any of his relatives.

The patient's obstetric history is as follows:—In about thirteen years she had a miscarriage, a girl, a miscarriage, three boys, a girl, twin girls, another girl, and an early miscarriage. This last was quickly followed by the pregnancy now in question.

Pregnancy progressed favourably during the early months; but from mid-term onwards there was more abdominal enlargement and more discomfort than usual. At about the thirtieth week the abdomen was so distended that she began to have difficulty in getting about, and became very breathless. A series of attacks of vomiting completed her discomfort and drove her to bed. We saw her one Wednesday afternoon when she had been unable to retain food or to sleep for almost 48 hours. There was very considerable dyspnœa, and the pulse was very rapid and feeble. The circumference of the body was 48 inches, and when the patient lay on her back the abdominal tumour bulged over and lay on the bed beside her at one side or the other. A small area in each flank was resonant on percussion, and a narrow resonant band ran round the lateral and upper limits of the tumour, which reached within an inch of the ensiform cartilage. The tumour itself was absolutely dull, and a perfect thrill was transmitted through it in every direction. No solid structures could be felt. No fœtal heart-sounds were audible, nor could fœtal movements be felt. There were no rhythmical contractions. The abdominal physical signs were identical with those given by an enormous parovarian cyst, and did not in any way suggest the pregnant uterus. *Per vaginam*, the physical signs were typical of pregnancy in a multipara nearing the end of gestation. The os admitted one finger; bulging membranes could be felt, but no presenting part of a fœtus.

The case was clearly one of hydramnios, which had somewhat suddenly reached a stage demanding active interference. At 9 o'clock the same evening we gave an antiseptic douche, and then punctured the presenting membranes with a tubular instrument and drew off two quarts of liquor amnii. The flow then ceased completely, and on withdrawing the instrument a fœtal breech was felt through the os.

Examination of the abdomen now revealed a resonant band, some 2 inches wide, representing the transverse colon, and the clear area in each flank was also increased. The uterus, however, still exactly resembled a parovarian cyst. It was therefore clear that it contained more than one amniotic sac, since fluid remained after one sac had been emptied of its two quarts of liquor amnii. The patient was much relieved, and we determined to do nothing more that night. Next morning (Thursday) the patient was very much better. We did not see any prospect of a favourable continuance of pregnancy; on the other hand we wished to avoid vaginal interference. Further, the uterus had been so paralysed by over-distension that we were anxious to see some evidence of returning muscular power before taking any steps towards emptying the organ. We therefore decided to give quinine, and the patient had 16 grains of the sulphate in four doses during the day. That evening (Thursday), 24 hours after the withdrawal of liquor amnii, she was able to sleep and to take nourishment. The vomiting and dyspnœa had not returned, and the pulse was slower. There was no sign or symptom of labour-pains. No vaginal examination was made, and we went away remarking that the 16 grains of quinine had failed to improve the uterine activity.

Six hours later, namely, at 3 a.m. on Friday, we were sent for, and arrived to find a fœtus in process of expulsion. This was the one whose amniotic sac had been drained 30 hours before, and it was expelled without fluid. The hand passed into the uterus now encountered very tough bulging membranes. These being ruptured with difficulty, a gush of liquor amnii filled the bed and covered the floor, a couple of quarts being easily collected. A second fœtus was then readily delivered by the feet. Another amniotic sac was next opened, with another flood of waters and a third fœtus. The process was repeated yet again, and when the fourth child was safely extracted, the bed and the floor were simply drenched. The uterus contracted well, and there was practically no bleeding. An enormous placental mass came away easily, and the patient did not from that moment present any feature of interest.

The children were all living, but were very small, ranging from 18 ounces to 36 ounces in weight. They all died during the 24 hours following their birth. There were two males and two females. The secundines consisted of one small placenta, connected by membranes with the other three, which were united into a single mass. There were four complete amniotic sacs, each having a complete chorionic covering; that is to say, on examining the septum dividing one sac from the next, it was seen to consist of four layers—amnion, chorion, chorion, amnion—all easily separable from one another.

Apart from its interest as an instance of quadruplets, this case was a nice example of hydramnios gradually advancing until at last acute symptoms demanded active interference. From the therapeutic point of view, also, it may be regarded as an illustration of the value of large doses of quinine in obstetric practice. It may be argued that the case would have ended in the same way had no quinine been given; but a very considerable personal experience of inertia uteri treated with this drug would suggest that this was one of those cases in which absolutely physiological uterine action begins when 12 or 16 grains of quinine sulphate have been given within about six hours. The dilatation of the cervix under these circumstances is often unconscious, and the administrator is surprised on being summoned again, after another six hours or so, to find delivery imminent or complete. It is difficult to believe that in this case the enormously-distended and paralysed uterus would have contracted so well after labour had Nature been quite unaided by the quinine.

PLACENTA PRÆVIA WITH MARGINAL INSERTION, PRESENTATION, AND PROLAPSE OF THE CORD.

Case II.—Mrs. Y. is a healthy woman, 40 years of age. She has had several previous pregnancies, but these all ended normally, and her obstetric history is of no interest in the present connection. She went into labour at term after an ordinary pregnancy, and sent for her doctor some hours later. There was then considerable hæmorrhage, and the membranes were ruptured before his arrival. On making an examination he found a loop of the cord lying outside the vulva. An hour later abdominal palpation revealed the head still disengaged and above the brim. The vagina was full of umbilical cord, and it was impossible to get this back into the uterus even with the patient in the genu-pectoral posture. We then gave chloroform, and pulled the coils of cord outside the vulva, so as to make room for the hand in the vagina. One end of the cord could now be traced up into the uterus, while the other was found to end in the margin of the placenta just above the still incompletely dilated os uteri. The margin of the placenta was separated from the uterine wall for about 2 inches.

The condition of things now became clear. There was marginal placenta prævia, the placenta being of the "battledore" variety (marginal insertion of the cord). The cord was inserted into that part of the margin of the placenta which was nearest to the os uteri. When labour began, the prævial portion of the placenta was separated, with a certain amount of unavoidable hæmorrhage. As soon as the cervix opened there was presentation of the cord, this also being unavoidable owing to the insertion of the cord and the prævial position of that part of the placenta into which it was inserted. Prolapse of the cord followed when the membranes ruptured. Owing to the position of part of the placenta and of the cord within the lower uterine segment, the head was unable to engage, and this doubtless saved the child's life by preventing compression of the cord. As soon as the circumstances became clear, we saw that classical treatment by reposition of the cord was impossible. We therefore quickly completed the dilatation of the cervix with the hand, applied the axis-traction forceps without waiting for the head to engage, and delivered the child quickly enough to avoid fatal compression of the cord. It cried lustily at once, and the placenta followed without any difficulty.

From this case it would appear that prolapse of the cord, like hæmorrhage, may be either "accidental" or "unavoidable."

A NOTE ON THE BACTERIOLOGICAL DIAGNOSIS OF DIPHTHERIA.

BY GEORGE NEWMAN, M.D.,

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SINCE 1894 the classification of diphtheria-bacilli receiving general acceptance has been that of Park and Beebe[1] who suggested three provisional groups, as follows :—

1. The virulent diphtheria-bacillus of Klebs-Löffler.
2. The non-virulent diphtheria-bacillus.
3. The Hoffmann bacillus.

Since that time bacteriologists engaged in the study of the subject have been occupied in two chief lines of research, namely, (*a*) the relationship existing between these three groups, and (*b*) the presence or absence of members of these groups in the throats of diseased and healthy persons. As to the first point, the results at present attained are by no means conclusive. Most bacteriologists hold that the non-virulent bacillus and the Hoffmann bacillus are incapable of producing diphtheria, although there is not wanting evidence to show that alone or in association with other organisms they may create or aggravate pathological conditions in the throat.[2] This, however, requires much confirmation before being generally accepted.

At present divergent views are held as to the Hoffmann bacillus. (*a*) Some authorities claim that it is a distinct organism, differing fundamentally from the true diphtheria-bacillus, devoid of virulence and producing no toxines, widely distributed in nature and as common in healthy throats as in diseased.[3] (*b*) Others hold that the Hoffmann bacillus is non-virulent, but is a modified form of the Klebs-Löffler bacillus. They claim that it resembles the latter in almost all essential characters with the exception of virulence ; and as stated above there are those who hold that it possesses certain pathogenic properties though in a much less degree than the

Klebs-Löffler bacillus. These divergences become the more complex in view of the claim made by Hewlett and Knight,[4] and Richmond and Salter[5] that the non-virulent Hoffmann bacillus may be transformed into the virulent Klebs-Löffler *B. diphtheriæ*. The first-named workers cultivated the Hoffmann bacillus on blood-serum through a number of generations, and Richmond and Salter passed it through susceptible birds, and were thus able to exalt its virulence. The converse transformation has also been reported by Hewlett and Knight, namely, that 48-hour broth-cultures of the Klebs-Löffler bacillus when incubated at 45° C. for 4, 6, 17, and 24 hours respectively became apparently identical with the non-virulent Hoffmann bacillus. Roux and Yersin also produced an attenuation of the virulence of the Klebs-Löffler bacillus by various methods, and the result was the non-virulent form of that organism. But the converse transformation was not obtained.

These experiments are full of interest, but before accepting the conclusion that the Hoffmann bacillus can be changed into the true diphtheria-bacillus and thus become a factor in the spread of diphtheria a great deal more evidence is required. Hence it may be taken for granted that the general view at present held is that for all practical purposes the presence of the Klebs-Löffler bacillus is to be looked upon as necessary to the occurrence of diphtheria, but that the presence of non-virulent forms simulating the *B. diphtheriæ* should put the physician on his guard as to the possible infectivity of such a throat.

In passing it may be said that the differentiation between the true *B. diphtheriæ* and the organisms which are allied to it is now reasonably reliable and practicable, for whilst the characters of the allies are by no means uniformly present, the chief characters of the Klebs-Löffler organism are typical and more or less invariable. During recent years a great deal of attention has been paid to the differential features of this bacillus, and as a result of these researches it is now generally held that the following four points are sufficient, taken together, to demonstrate that the organism in question is a Klebs-Löffler bacillus:—(1) The macroscopic and microscopic appearance of the culture-growth on blood-serum; (2) the staining-properties of the bacillus, and in particular its character when treated by Neisser's stain; (3) the reaction shown by a subculture in neutral or slightly alkaline litmus-dextrine-broth after 48 hours at 37° C., which is rendered acid by Klebs-Löffler bacillus; and (4) its pathogenicity, tested by inoculation in guinea-pigs.[6] In actual laboratory practice the tests (1) and (2) are those relied upon for giving at all events a provisional diagnosis. Opinions vary as to the value of Neisser's stain for diagnostic purposes, but in general it may be said that, whilst it cannot be relied upon for differentiating the virulent Klebs-Löffler from the non-virulent form, it is a test by which it is possible to separate the Hoffmann bacillus from the other two.[7]

The next point to consider is the relative occurrence of the true *B. diphtheriæ* in the throats of persons showing more or less clinical sign of diphtheria, in the throats of healthy persons who have come into contact with cases of diphtheria, and in the throats of healthy persons who have, as far as is known, not come into contact with the disease.

In respect to notified cases Graham Smith[8] has pointed out that of nearly 27,000 cases of certified diphtheria about 72 per cent. give a positive result on bacteriological examination These cases were observed in various countries and places, which introduces a possible fallacy in respect of the personal equation. For instance Cobbett[9] in two outbreaks at Cambridge reported that in 57 per cent. and 66 per cent. respectively of the notified cases it was found possible to demonstrate the presence of the *B. diphtheriæ;* but in some German investigations the percentage reached 97. Such figures must be judged broadly, and to be of scientific value the conditions and circumstances surrounding the cases must be considered in respect to each return. It is evident that a very slight difference in circumstances might materially affect the result.

What is perhaps of more moment from the diagnostic point of view is the percentage of doubtful cases in which it has been found possible to demonstrate the bacillus. It is well known that it is the practice of many local authorities, especially in large urban districts, to provide bacteriological examination of "swabs" sent to them by medical men practising in the district. A study of the returns of such examinations will furnish some idea on the point under consideration. In the following metropolitan districts the returns for 1902 were as follows :—

The presence or absence of *B. diphtheriæ* in Doubtful Cases in some Metropolitan Districts in 1902 :—

Metropolitan Borough.	Total.	Positive.	Percentage of Positive.
Westminster - - -	179	52	29·0
St. Pancras - - - -	175	49	28·0
Islington - - - -	122	50	40·9
Finsbury - - - -	112	27	24·1
Woolwich - - - -	97	27	27·8
Lambeth - - - -	154	16	10·3
Poplar - - - -	137	48	35·0
Hackney - - - -	25	11	44·0
Stoke Newington - - -	29	10	34·4
Greenwich - - - -	60	15	25·0
Paddington - - - -	36	15	41·6
Camberwell - - -	234	111	47·4
Wandsworth - - -	72	29	40·2
Fulham - - - -	236	61	25·8
Holborn - - - -	17	8	47·0

These figures from 12 districts give an average of 32·7 percentage of positive results. In 1901, for seven of the above districts, of which the returns are obtainable, the percentage of positive cases was 34·6. In Liverpool in 1902 the percentage of positive cases was 29·3 (Hope). In Edinburgh it was 22·4 in the same year (Littlejohn).*

It is now generally recognised that healthy persons after coming into contact with diphtheria-patients may themselves harbour the virulent bacillus. The actual percentage of such occurrences varies widely,[10] and is obviously dependent upon many external conditions. In families where a case of clinical diphtheria exists there may be from 50 per cent. (Park and Beebe) to 100 per cent. (Cobbett). In schools and institutions the percentage is of course lower. Goadby found it to be 32 per cent. in the Poplar Union Schools ;[11] Aaser in a certain soldiers' barracks showed that it was 19 per cent. ;[12] and Denny found in a truant school that it was 11 per cent.[13] The

* In the West Riding of Yorkshire, during 1902, 1,486 specimens were examined and 736 (or 49·5 per cent.) were positive (Cammidge).

present writer had the opportunity in 1901 of observing an instance of a healthy person conveying virulent diphtheria-bacilli to the throat of a child who suffered from clinical diphtheria; from this patient several healthy persons again conveyed the organism to others, who in their turn suffered from diphtheria. In all, 15 persons came into contact with the child; five remained well and their throats were free from the bacillus; 6 others remained well but carried the organism in their throats; and 4 contracted clinical diphtheria from this conveyance of the bacillus.[14] In all these 15 persons the throat was bacteriologically examined.

During the year 1902 Dr. R. K. Brown, of Bermondsey, visited 102 families in which diphtheria had occurred. Examinations were made of the members of the family who were in immediate contact with the patient, and these examinations were practically confined to "contacts" of school age, *i.e.*, up to 15 years of age. The number of examinations made was 259, and swabs were taken from the throats, and, in a few cases, from the noses of those examined. In 32 specimens, or 12·4 per cent. of the examinations, Klebs-Löffler bacilli, which constitute the immediate infective agent of diphtheria, were found: 16 of these cases, or 50 per cent., later developed the clinical symptoms of diphtheria, and were removed to hospital. Those who had bacilli in their throats, but did not develop the clinical symptoms of diphtheria, were kept isolated, as far as possible, until they were considered to be free from infection, and were advised to go to their medical attendant for an antiseptic gargle. These latter did not give rise to any further cases in the household. In all cases of children in which diphtheria-bacilli were found, the parents were immediately notified that their child harboured diphtheria-bacilli in its throat, and might either develop diphtheria later, or be a source of infection to others and were advised to keep the child in question under the close observation of their medical attendant. By this method there is no doubt the 16 cases notified were placed under treatment and isolated much earlier than they would otherwise have been.[15]

Lastly, it has been shown that healthy persons who, as far as known, have not been in contact with diphtheria may harbour the *B. diphtheriæ* in their throats. Denny examined 235 healthy individuals, of whom 216 were children, and found one case only. Kober examined 600 healthy children, and found diphtheria-bacilli in 15 cases; Biggs met with 8 such cases out of 330 healthy persons; and the Committee of the Massachusetts Association of Boards of Health report that 1–2 per cent. of healthy persons amongst the general public are infected with diphtheria-bacilli. Only 17 per cent. of such bacilli appear, however, to be virulent. Hewlett and Murray have surpassed other investigators in this direction, and state that one in seven of all sick children in the Victoria Hospital for Children have virulent diphtheria-bacilli in their throats.[16] The suggestion that this percentage of 15 is one commonly obtaining amongst normal children is surely unsupportable. Indeed, for a children's general hospital the return is probably exceptionally high. In all such returns, moreover, it is desirable to state particulars of possible infection and some history of each case. Probably, in general, it may be said that 1–2 per cent. of healthy persons harbour diphtheria-bacilli in their throats, and it is not improbable that unconsciously such persons have been in contact with direct or indirect infection from actual cases of the disease.

In view of the above facts, the question naturally arises, what is the best practical line of action? To which the answer would seem to be :—

(*a.*) In the presence of a case of doubtful clinical diphtheria bacteriological examination of the throat should be made. If the result be positive the case should be notified, and where home isolation is impossible or impracticable the case should be isolated in hospital. If the result be negative a second and, if necessary, a third, examination should be made.

(*b.*) Persons isolated in hospital should not, as a rule, be discharged until their throats are clear, however convalescent they may otherwise appear to be. Two or more negative examinations are usually necessary.

(*c.*) In the event of clinical diphtheria, supported or unsupported by bacteriological examination, the throats of other persons in the house should be examined for the *B. diphtheriæ*. If the result be positive, such persons, if not suffering from clinical diphtheria, should be isolated at home until their throats are clear. If the result be negative, their throats should be reëxamined in due course.

(*d.*) It is unnecessary to add that the early use of antitoxine, both as a therapeutic and as a prophylactic agent, is to be recommended.[*]

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[*] The value of antitoxine treatment in diphtheria is clearly demonstrated in the returns of the Metropolitan Asylums Board. In 1894, 3,042 patients of all ages were treated in the Board's hospitals without antitoxine; 902 died, yielding a mortality of 29·6 per cent.

In 1895 the antitoxic-serum treatment was inaugurated; 3,529 cases of diphtheria were treated, and 729 died, yielding a mortality of 22·5 per cent. Hence, in the first year there was a fall in mortality of 7·1 per cent.

In 1901, 6,499 patients suffering from diphtheria were treated with antitoxine in the Board's hospitals; 817 died, yielding a mortality of 12·5 per cent. There has, therefore, been a fall in mortality percentage from 29·6 in 1894, without antitoxine, to 12·5 in 1901, with antitoxine. In other respects the treatment has been substantially the same. In 1902, 7,707 cases of diphtheria were treated with antitoxine, and 739 died, yielding a mortality of 11·04 per cent. (as against 29·6 per cent. in 1894).

At the Metropolitan Asylums Board Brook Hospital in 1901, 723 cases of diphtheria were treated with antitoxine, and 78 died, yielding a mortality per cent. of 10·79. The antitoxine treatment was applied in each of these cases, but in some it was possible to begin on the first day of the disease, in others on the second, and so on. The paramount importance of administration at the earliest possible moment is seen in the result. The percentage mortality of the first-day cases was 0·0; of the second-day, 4·1; of the third-day, 11·9; of the fourth-day, 12·4; and of the fifth and subsequent days, 16·6. For five consecutive years there has not been a death at this hospital among the cases that came under treatment on the first day of the disease. Even more striking evidence of exactly the same nature has just been reported in Massachusetts as the result of seven years' experience with antitoxine.

REFERENCES.

[1] New York *Medical Record*, September, 1894.
[2] *Jour. of State Medicine*, October, 1903, p. 612 (J. C. Heaven).
[3] *Ibid.*, p. 612 (Cobbett).
[4] *Trans. Brit. Inst. Preventive Medicine*, 1897.
[5] *Guy's Hospital Reports*, vol. liii., and *Trans. Jenner Institute* (second series), 1899.
[6] *Medical Officer's Report to Local Government Board*, 1901-02 (M. H. Gordon), p. 418, *et seq*.
[7] *Medical Supplement of First Annual Report of Hospitals Committee to Metropolitan Asylums Board*, 1900 (W. T. Gordon Pugh), p. 136, *et seq*
[8] *Jour. of Hygiene*, April, 1903, p. 217.
[9] *Ibid.*, 1901, vol i.
[10] *Ibid.*, October, 1903, p. 218 (Graham Smith), and W. T. G Pugh, *loc. cit.*
[11] *Trans. Epidem. Soc*, London, vol. xix.
[12] *Deutsch. Med. Woch.*, 1895.
[13] *Boston Med. and Surg. Jour.*, November, 1900.
[14] *Report on Health of Finsbury*, 1902, pp. 61–62.
[15] *Report on Sanitary Condition of Bermondsey*, 1902, p. 10.
[16] *Brit. Med. Jour.*, 1901, vol. i., 1474-5.

Notes by the Way.

The Courage of "King's."

We welcome the announcement recently made that it has been decided to move King's College Hospital from its present site near the Strand to some position, yet to be determined, on the other side of the river. It has long been evident that in the former region there was a plethora of hospitals, Charing Cross Hospital, King's, and the Middlesex being all within a short radius. It was, therefore, highly desirable that one of these institutions should remove to some less central spot. On the south side of the river there are large areas inhabited by a poor population who have no available hospital assistance, and it will not be difficult to find a suitable site for the reërection of "King's." Yet it must have needed much courage and self-denial, as well as wisdom, on the part of the staff and governing body to make up their minds to the final plunge. Courage is not so invariable an attribute of public bodies that we can take the exercise of it for granted, and we very heartily congratulate those responsible for the destinies of King's College Hospital on the step which they have taken. We hope that the wisdom of the course adopted will appeal to the public, and ensure that sufficient funds are forthcoming to enable the transfer to be effected without even temporary loss of efficiency, and that the new hospital, when it is erected, will worthily uphold the traditions of the old. We shall watch with interest to see whether the authorities who preside over the administration of the great central funds collected for the hospitals will give effective support to the first institution which has adopted the course which they have urged in the past, and which they should feel bound to encourage. Any failure on their part to render substantial aid will fatally, though justly, diminish their future powers of influencing hospital policy.

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The Army Medical Service as a Career.

THE changes which have recently been made in the conditions of the Royal Army Medical Corps cannot be too widely known to students of medicine. Not long ago it was exceptional for those who had had successful careers at our medical schools to enter for the army examination, and the authorities did nothing to encourage, if they did not actively discourage, students who exhibited any leaning in this direction. At that time there were many drawbacks to the position of a military surgeon which acted as deterrents to enquiring candidates. We are happy to feel assured that these defects in the conditions of service have now been removed, and both the army and the medical profession will benefit by the change. Indeed, the attractions now offered to an ambitious young man at the outset of his medical career must make the Army appeal forcibly to the imagination. In the future it would seem that we shall have to take care lest all our most accomplished students are absorbed by the Royal Army Medical Corps, in which the natural attractions of fixed remuneration and the chance of seeing the world are no longer counterbalanced by deficient pay and unsatisfactory status. It is now possible for the Army surgeon to live on his pay with reasonable comfort. He is entitled to retire with a gratuity of £1,000 at the end of 9 years' service, and the amount rises to £2,500 at the end of 18 years. After 20 years on the active list, a Major or Lieutenant-Colonel in the Royal Army Medical Corps may retire on a pension of £1 a day, or after 30 years on £455 per annum. Higher posts carry larger pensions. There is every reason to hope that promotion will be to a great extent the reward of merit, so that a hard-working officer will be sure to find his work recognised. Further, study-leave will be more freely granted, and opportunities thus afforded to the members of the Royal Army Medical Corps of keeping themselves well acquainted with the advances of medical knowledge.

It used at one time unfortunately to be the fashion among the ignorant to decry the Army doctors as less efficient exponents of their art than their civilian brethren. We believe that the reproach was always unjust, and it would be possible to point in disproof to admirable work in the sphere of scientific research done by members of the Army Medical Service in the past. We hope that as a result of the reorganisation which has been effected we may see the end of that tendency to antagonism between the civilian and the military branches of our profession. Both sides may have been at fault in the past, but much will have been accomplished for the general good, if it comes to be recognised that it is the worst possible "form" to attack one's own profession publicly, still more to make charges against a whole class without specifying individual faults, as has sometimes been done, in the shape of sweeping charges to which no kind of answer can, in the nature of things, be attempted. Unity is strength, in this as in other instances; and if all branches of the profession will unite to exercise a little mutual charity, they will find that the public at large will have a higher opinion of medical men individually and collectively.

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Patent Medicines and Public Health.

We live in a free country and tend to pride ourselves on the fact, though it is noticeable that the phrase is usually quoted when we have to point to some glaring defect in our institutions, by which in some particular instance liberty is allowed to border on licence. Englishmen of more than usually philosophical bent may at times be induced to admit that there are some things in which foreign countries are wiser than ourselves, and it may not be too unpatriotic to enquire—enquiries are the fashion—whether the recent ordinance in Germany against the sale of secret remedies is an instance of effete despotism, or rather an enlightened enactment in the interest of public health. Is it really a public advantage that any manufacturer, with or without an elementary knowledge of drugs, should be able to concoct a mixture of unknown ingredients, and advertise it broadcast as a cure for the stomach-ache and the thousand natural shocks that flesh is heir to? True, we are equally free to disbelieve the alluring posters' tale, but how are the unsophisticated to know when to disbelieve? Is not every panacea backed by an array of certificates from clergymen and others lauding its efficacy? Human nature tends to believe anything which is asserted loudly and continuously. Hence the enormous consumption of nostrums by our countrymen; and we believe still more is absorbed per head in the United

States. We may well ask, is our boasted freedom in this respect good? That many cases of ill-health may be attributed directly to the habit of self-doctoring is undeniable. What dermatologist has not seen cases of lesions caused or aggravated by dosing with secret remedies containing iodide of potassium? What medical man has not had to treat confirmed constipation due to prolonged abuse of purgative drugs? Cases of serious disease which have passed the stage at which they were curable, while the sufferer was swallowing first one and then another infallible panacea, are of only too frequent occurrence. Are we sure that we manage things so very much better in this respect than they do in Germany?

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Infant Foods.

BUT even granted that adults should be left free to swallow any noxious substance which they may choose to take, in utter ignorance of its nature or action; in the case of infants the matter stands on a different footing. They cannot choose what they will have to eat, and too often they get that which is least suitable to their needs. Is it right that anyone should be allowed to concoct a starchy food or biscuit, and advertise it as a fitting food for infants from the day of their birth? We believe that more deaths may be traced to the use of proprietary foods among the nurselings of the poor than to many an epidemic. Yet no one makes any endeavour to abate the ill. We appoint commissions to enquire into the cause of the physical degeneration of the children of our great cities, but the advertisement of poisonous foods for babies goes on unchecked. The words are none too strong, for poisonous to babies they undoubtedly are when they contain materials quite unsuitable to their tender stomachs. How many stunted, rickety, ailing children who attend any out-patient department at a children's hospital owe their troubles to patent foods, given by their ignorant mothers on the strength of advertisements? And how are these mothers to know that the specious posters only invite them to poison their infants and perhaps cripple them for life, when they offer such alluring pictures of health and plumpness as baits? Well may we say, "O Liberty, Liberty, how many crimes are committed in thy name!"

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Gastric Luxuries and Gastric Laziness.

We live in an age of patent foods, all calculated to save the stomach from having to perform its proper work. By nature it is a long-suffering organ, but it may be tried beyond its endurance. Then disagreeable symptoms occur. It may have been overcrowded with rich food, when all it asks is to be relieved of its load, to have a short rest and to start afresh on physiological lines. It may have been called upon to do its work with an insufficient supply of blood, either owing to general anæmia or to mental work being carried on at the time when digestion should have been proceeding alone; or some other similar cause of temporary derangement may have been at fault. At once the cry is raised, "The stomach is out of order." Its work must be done for it; we must give peptonised food. What is the result? Instead of being encouraged to digest, the poor organ is kept in such a condition that it has not a chance of carrying on its functions. It is encouraged to forget its duties, and to pass its time in idleness, while it is soothed with bismuth or morphine or cocaïne, the better to complete its enervation. And so dyspepsia becomes chronic, and peptonised food a necessity. Whereas if the stomach had been given a short rest, and perhaps been encouraged by a little commonplace gentian and soda, it would have come up smiling, fit to digest good wholesome meat and pudding.

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Extracts.

Just as our minds are fed on tit-bits, on extracts from far and near in the way of mental pabulum, so we are encouraged to swallow an endless amount of animal and vegetable extracts under the delusion that they contain a concentrated form of nourishment, which will make the puniest town-dweller into a Hercules without delay. Babies are fed on them, weakly youths are dosed with them, dyspeptics absorb them for their lunch. And how much nutriment is there in an average meat-extract? It is generally made by some process which involves the heating of the meat to a point above that at which the albuminous constituents coagulate; and all that it contains is the extractives —a useful form of stimulant no doubt in some cases of disease, an agreeable form of flavouring agent in cookery, but as nourishment a cupful is about equal to a tumbler of water with a teaspoonful of milk in it. *Vanitas vanitatum, omnia vanitas!*

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The Bacillus of Syphilis.

A YEAR or two ago a communication appeared, from Drs. De Lisle and Jullien, announcing the discovery of a bacillus of syphilis. A further report has lately been published by Dr. De Lisle on this organism (*American Medicine*, September 19, 1903) in which additional details are given. The bacillus is a motile organism, which rapidly breaks up on withdrawal from the body of its host, as the blood in which it is contained coagulates. It has been cultivated, however, by mixing the plasma of a syphilitic subject (not the serum) with broth, and incubating portions of the fluid in collodion capsules in the peritoneal cavities of guinea-pigs. Subsequently the organisms will grow on ordinary broth or gelatine. The bacilli are agglutinated by the blood of syphilitic patients, but not by that of healthy persons. A special immune-body or copula is formed in the serum of animals inoculated with the blood of syphilitics which is capable of causing destruction of the bacilli in the presence of an alexine or complement contained in normal blood. These results are very interesting, and afford at least *primâ facie* evidence that the bacilli are the causal organisms of the disease.

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Dilatation or Dilation?

"A LITTLE knowledge is a dangerous thing." Our learned contemporary, *American Medicine*, devotes a column to endeavouring to prove that the proper word for "the act of dilating" is "dilation," and not "dilatation." Had this excellent journal been content to base its argument on its statement that "We live in a grammarless Eden. Philologic barbarism is the substance of English or any other modern derivative language," we should not have felt bound to notice the contention, merely making a mental note that no doubt the American dialect was meant when the English language was said to be invariably ungrammatical. But *American Medicine* proceeds to justify its preference for the mistaken form "dilation" on the ground that the past participle of the Latin verb *dilatare* is *dilatus*. With all due deference, we would point out that *dilatus* is the participle of *differo*, which means to "I put off," and not "I dilate," the participle of *dilatare* being *dilatatus*. The further contention that "dilation" must be right on the analogy of "collation" and "elation" shows still further confusion, since these latter terms are derived directly from *confero* and *effero* respectively, and therefore stand on a different footing from "dilatation," which is not immediately derived from *differo*. *American Medicine* seems to doubt the wisdom of devoting a column to a discussion of the rights and wrongs of a mere etymological question; we are almost inclined to think the doubt justified in view of the argument.

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Treatment of Uterine Fibroids.

THE paper in our present issue by Mr. Alban Doran on the treatment of fibroid tumours of the uterus contains a practical and impartial appreciation of the advantages and disadvantages respectively attaching to operative and medicinal methods. On the one hand, we see many cases in which the mere existence of a fibroid is not associated with any considerable degree of ill-health; in which there is, perhaps, a rather greater loss of blood at the monthly periods than would otherwise occur, but really severe hæmorrhage is not met with, or if it occur, is easily controlled by appropriate remedies. To advise removal of the growth under such circumstances appears, not only unnecessary, but actually improper. On the other hand, cases occur in which the patient's life is made a burden by the growth and the evils dependent upon it. In such a case we need not hesitate to recommend removal; indeed it is cruel to dissuade such a woman from her natural desire to have some operation performed. Yet we have known of cases in which operation was put off for years, during which the sufferer was condemned to a life of hopeless invalidism, only to have to undergo hysterectomy at last when the symptoms had increased beyond endurance, and when the patient was in a much weaker condition and less able to stand the shock than if she had been operated on at first. Mr. Doran points out that although a fibroid which is causing no serious disturbance of health should not be removed on the mere chance that it may some day become troublesome, yet it must be carefully watched. If, on the other hand, the symptoms are so marked as to interfere with the patient's enjoyment of life, we may advise removal of the uterus with good hope of success, now that the technique of this operation has been so far perfected that in skilful hands the risk attaching to it is no longer alarming.

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The Age-Incidence of Tuberculosis.

IT is generally considered that the risk of tuberculosis diminishes rapidly after the age of about thirty years—that it is, in fact, a disease of the young. The paper by Dr. Lister on the Relation of Tuberculosis to Life-Insurance, which will be found on another page of our present issue, tends to contradict this assertion. It seems that in actual fact the deaths from phthisis form a very constant proportion of the whole number of deaths which occur at each successive decade, and this constancy is attributable to the fact that there are three factors at work in the incidence of tuberculosis—heredity, acquired predisposition, and infection. The hereditary predisposition gradually diminishes as a factor in the death-rate, those who possess it tending to be killed off in early life, while the acquired liability becomes a gradually-increasing risk, the opportunities for infection meanwhile remaining practically equal throughout life. It is true that there is a slight falling off in the number of deaths from phthisis at the extreme end of life, which Dr. Lister attributes to failure to recognise the tubercular nature of many of the cases of lung-disease seen among the old, in which the clinical symptoms may be indistinguishable from those of simple bronchitis and emphysema; but even thus the proportion borne by the tubercular cases to the whole number of deaths at every age remains when put into diagrammatic form a very even band.

Reviews of Books.

A Text-book of Obstetrics for Practitioners and Students. Edited by R. C. NORRIS, M.D., and R. L. DICKINSON, M.D. With nearly 900 illustrations. Two vols. Second edition. 1902. London and Philadelphia: W. B. Saunders & Co.

THIS second edition is published in two volumes. A number of illustrations have been replaced by others and some new ones have been added. The chapters by the late Dr. Etheridge have been re-written by Dr. J. Clarence Webster, while Dr. Norris has revised those from the pens of the late Drs. Parvin and Earle. The first volume contains seventy pages of anatomy, but the physiology of the female generative organs is compressed into three brief pages on ovulation and menstruation. The next sixty-four pages are devoted to human embryology, well written and beautifully illustrated. The bulk of this embryology is, however, entirely out of place in a text-book of midwifery. The development of the brain and sense-organs, for instance, has no connection whatever with the subject in hand. There is much repetition, but this is doubtless unavoidable in a work written by many authors. Still, the excessive overlapping which occurs in the chapters on Physiology of Pregnancy and Diagnosis of Pregnancy should have been avoided in a second edition. The arrangement is also very faulty in places: thus under the heading "The Urinary Organs during Pregnancy" we find "Suppurating Hydatid of the Abdomen," "Peritonitis during Pregnancy," "Concealed Accidental Hæmorrhage," "The Posture and Bearing of the Pregnant Woman," "Relaxation of the Pelvic Ligaments," "The Toxæmia of Pregnancy," all the nervous diseases of pregnancy and a number of other subjects. The latter portion of the first volume consists mainly of "mechanism," and the authors have not succeeded in making their mathematical or mechanical midwifery very attractive or convincing. The second volume first deals with "Dystocia"; and it is curious to note that under this heading accidental hæmorrhage is described for the second time in the book. Placenta prævia, post-partum hæmorrhage, rupture of the uterus, hyperemesis, and a number of other "diseases of the mother" are also mentioned under "Dystocia." But what is the use of having a word, if you cannot change its meaning and connotation? The section dealing with "Puerperium" is better than some. The pages devoted to "new-born infants" are good, but the best part of the book is the last section, which deals with obstetric surgery. The Americans, on the whole, are much better at surgery than they are at obstetrics, or at writing and spelling and book-making. After keeping the volumes beside us for several days and turning to them for information on various points, the conclusion is that there are several text-books of midwifery by single American authors which are more convenient works of reference than the present collaborative effort. The Art-editor, Dr. R. L. Dickinson, has done his work admirably, and is to be most heartily complimented upon it; and the publishers have done their part in their usual good style. There is an excellent index to each volume besides a general index; but the tables of contents are worse than useless.

Handbook of Obstetric Nursing. By FRANCIS W. N. HAULTAIN, M.D., F.R.C.P. (Ed.), and JAMES HAIG FERGUSON, M.D., F.R.C.P. (Ed.), M.R.C.S. (Eng.). Edinburgh and London: Young J. Pentland. 1902. Price 5s.

THE fact that this handbook has already reached a fourth edition would alone prove its usefulness sufficiently as a book of reference for the advanced obstetric nurse. It is very clearly written, and has a large number of excellent illustrations, which are a great help to the memory. If a fifth edition of the book should be demanded, as we should think highly probable, we would suggest that the authors might, with advantage, give a somewhat clearer description of the so-called Crédé mode of expelling the placenta, so as to make it perfectly plain to a nurse not already familiar with the method. We should also like to see the great importance of prolonged and absolute rest after Phlegmasia alba dolens more emphatically impressed on the nurse. We can most cordially recommend this excellent handbook to all obstetric nurses who wish to have a thoroughly up-to-date guide in their work.

Aids to Gynæcology. By ALFRED S. GUBB, M.D. (Paris). M.R.C.S. (Eng.). London: Ballière, Tindall, and Cox. 1903. Price 2s. 6d.

THAT a fourth edition of this book has made its appearance bespeaks for it a certain degree of popularity. With regard to the legitimate use of "aids," it may be that some subjects lend themselves to this method of teaching, but if we may judge by the present work, Gynæcology certainly does not. There are many errors and discrepancies in the book:—the anatomy of the ovary is, to say the very least, misleading; the student will gather that the term mesometrium is synonymous with broad ligament; common and rare diseases are huddled together without distinction into tables, to be learned off by a novice in parrot-like fashion. Amongst the methods of treating pruritus vulvæ, excision of the clitoris is recommended; and in the treatment of retroflexion, we are told that ligatures may be passed through the cervix and then through the fundus, when they are to be drawn tight and so bring the uterus into anteflexion! For inversion we must amputate the fundus. Alexander's operation is lauded for procidentia uteri—a purpose for which it was never intended and for which we should hope it is never done. There are, indeed, many statements in this book which, if quoted, would be fatal to a candidate's chance of passing any examination, so that we are compelled to regard the word "aid" as a misnomer when applied to it.

Aids to Forensic Medicine and Toxicology. By WILLIAM MURRELL, M.D., F.R.C.P., Physician to, and Lecturer on Clinical Medicine in, the Westminster Hospital, &c. 6th edition. Pp. 122. London: Baillière, Tindall, and Cox. 1903. Price 2s. paper, 2s. 6d. cloth.

THIS little book evidently meets a need felt by medical students, and indeed it seems well calculated to act as an aid to getting up the subject of Forensic Medicine just before an examination, if taken, as recommended in the preface, "in small doses three or four days before going in." Under Forensic Medicine there is given an account of the commoner forms of insanity, and there is also a paragraph containing sound advice on dealing with some forms of blackmailing to which medical men are especially liable to fall victims. We regret, however, that some of the statements made are examples of deplorable taste, if not of absolute indecency.

The Diseases of Infancy and Childhood. For the use of Students and Practitioners of Medicine. By L. Emmett Holt, M.D., LL.D., Professor of Diseases of Children in the College of Physicians and Surgeons (Columbia University), New York, &c. With 225 illustrations, including nine coloured plates. Second edition, revised and enlarged. London: Henry Kimpton. 1903.

We cordially welcome the appearance of the second edition of Dr. Holt's well-known work on the Diseases of Children. It is in some ways the best book on the subject that we possess, and the present edition has been to a great extent re-written and thus brought up to date. It is profusely illustrated, and many of the pictures are very good. Naturally they are not all of equal merit. There is a useful coloured plate showing the appearance presented by Koplik's spots in measles, which may be commended to those who are not already familiar with this phenomenon. The plate showing the membrane in diphtheria is rather diagrammatic than exact in point of colouring. The common ailments of children are fully discussed in this book, and their pathology and morbid anatomy are also explained, drawings and photomicrographs of the latter being given. We doubt whether the condition known as Hodgkin's disease is fairly dismissed in half a page, while we find no reference to that curious malady affecting the joints in children, which has been fully described by Still, and is generally looked upon as corresponding with the rheumatoid arthritis of adults. The table of the dates of eruption of the milk-teeth seems to put their appearance later than is usually described, and we think seen, in this country, as a twelve-months' baby is here expected to have only six teeth, instead of nearly twice that number, as is usually taught. The subject of infant-feeding and of the various modifications of cow's milk for the purpose is very fully treated, the sections dealing with it having been entirely re-written. We would call the attention of practitioners in this country to the comparatively favourable prognosis given in this work with respect to cases of sarcoma of the kidney treated by operation, as we have seen such cases left to die without any attempt to save them, owing to the opinion that they were hopeless. Dr. Holt quotes statistics giving a recovery-rate of 33 per cent. after operation, and gives as illustration a case which recovered and was alive ten years afterwards. We heartily commend this book to all who are interested in the maladies of children.

System of Physiologic Therapeutics. Vols. V. and IX. Edited by SOLOMON SOLIS COHEN, A.M., M.D., Professor of Medicine and Therapeutics in the Philadelphia Polyclinic. Illustrated. London: Rebman, Limited. 1902.

THESE two volumes form part of a work which has at any rate the merit of being sufficiently comprehensive. Why the therapeutics considered in this system should be saddled with such an ugly name as "Physiologic" we fail to understand, unless it be that the well-recognised term "physiological" would be too commonplace for such advanced science as "Cromotherapy," "Heliotherapy," and "Dietotherapy," which, together with several other varieties of "therapy," are included in Vol. IX. Dr. Oliver Wendell Holmes once remarked that "quackery hobbles on two crutches, the tattle of women, and the certificates of clergymen"; possibly had he written in these days he might have added to these two crutches a third, namely *sesquipedalia verba.* True knowledge is not advanced, but hindered, by the invention of such scientific bombast as "cromotherapy," "psychrotherapy," and such-like terms. We can well imagine that some thoughtful readers, met on the threshold of these volumes by such titles as these, will turn back in fear lest quackery lurk within. But if they can get over this repugnance to these minor faults, they will find a wonderful, if somewhat motley, collection of facts accumulated in this system. Volume V. deals with the prophylaxis of disease, personal and civic hygiene, and the care of the skin. Under these headings almost every conceivable department of theoretical and practical medicine is lightly touched upon, from the most obscure problems of pathological chemistry and the various forms of anopheles up to the shape of boots and the advantages of shaving. There are many illustrations, some of which are excellent. In volume IX. the various "therapies" to which we have referred are described. The chapters on light-treatment are perhaps some of the most interesting. The sections on treatment by baths and natural springs and waters contain an enormous amount of information, but naturally refer chiefly to American resorts, although a praiseworthy effort is made to include also those of Europe. In both these volumes reference is facilitated by a capital index, which it must be confessed is much needed; for as each complete science and art of healing, as represented by "psychrotherapy," "acrato-thermal therapy," "hypodermoclysis," &c., claims as many diseases as possible, the study of the various methods of treating any one malady necessitates references not only to several parts of one volume, but also to many parts of several.

The Practitioner's Handbook of Diseases of the Ear and Naso-Pharynx. By H. MACNAUGHTON-JONES, W. R. H. STEWART, WILLIAM MILLIGAN, HERBERT TILLEY, AMBROSE BIRMINGHAM, and ROBERT DWYER JOYCE. With 182 illustrations and seven plates. Sixth edition. Demy 8vo. Pp. 368. London: Baillière, Tindall, and Cox. 1902. Price 10s. 6d. nett.

THIS is the sixth edition of a work which first saw the light in 1878. It contains, therefore, the ripe experience of the editor, Dr. Macnaughton-Jones, who is responsible for the chapters on examination, ætiology, symptomatology, therapeutics, the external ear, the internal ear, and on deaf mutism. These are all well and carefully written, and show the author's skill in technical details and his resourcefulness in treatment. For this last edition he has called to his aid a perfect galaxy of talent to assist in making up the 368 pages of which it is composed. No less than five other names appear on the title-page, and to these should be added that of Dr. Dudley Buxton, who has contributed a condensed chapter on the administration of anæsthetics in nasal and aural surgery. Professor Birmingham and Dr. Joyce are responsible for the applied anatomy of the ear. Mr. W. R. H. Stewart has written the chapter on the middle ear; and Dr. Milligan that on chronic suppurative middle-ear disease; while Dr. Tilley gives a good epitome of diseases of the nose in their relation to ear symptoms. This has lately been rather a vexed question amongst specialists, and we believe that a commission composed of members of the Laryngological and Otological Societies is at the present moment investigating it. Dr. Tilley's views on the relationship of nasal and aural affections are put forth clearly and judiciously. We notice that apparently he has entirely accepted the long discredited, but recently-revived, view of Woakes in regard to the ætiology of nasal polypi. On page 268 he writes: "They are the result of chronic inflammatory changes in the mucous membrane of the ethmoidal region, *due to an underlying inflammation of the ethmoid bone.*" (The italics are ours.) Now, while there is no denying that in a large number of cases of nasal polypi there is an accompanying osteitis of the subjacent ethmoid, it has not yet been definitely proved that this is the cause and not the consequence of the spread of superficial inflammation. Indeed such well-known pathologists and rhinologists as John Mackenzie and Hajek have carried out investigations which convince them that the inflammation commences on the surface and spreads inwards. Dr. Tilley's statement is, therefore, somewhat "too previous," and particularly so in a book intended for practitioners who, if they accepted this teaching, would consider themselves justified in attacking the ethmoid bone, as the *fons et origo* in all cases of polypi. In Fig. 142 there is a mistake in the description: as it is a drawing of a reversed image, the terms "right" and "left" should be transposed.

In Mr. Stewart's chapter we notice that our old friends "cold" and "chills" are made responsible for much that is nowadays more correctly attributed to microbial infection. We wonder on what pathological principles he states that change of air is "imperatively required" should an attack of myringitis become intermittent (page 155). A remarkable oversight has been made in the same chapter in leaving out all reference to any antiseptic precautions before or after paracentesis of the tympanic membrane. To all who desire to carry out this operation "safely and efficiently"—as the writer wisely recommends—we should have thought that scrupulous antiseptic precautions were of the first importance.

Dr. Milligan's chapter shows considerable knowledge of the subject, though much of it appears to us disproportionately planned for a practitioner's handbook. Thus, only four pages are devoted to opening the mastoid for recent, acute suppuration, while ten are given up to the same operation for chronic otorrhœa. While agreeing that the more complicated nature of the latter operation requires more space for dealing with it, we think that the former should have received fuller recognition. The practitioner may be frequently called on to open the mastoid antrum in acute disease. The difficult, tedious, and sometimes uncertain operations on the mastoid for chronic otorrhœa are often "radical" only in name and not in result. They are rarely called for in good-class general practice, and the practitioner cannot afford the time to carry them out satisfactorily amongst the poor, who, besides, are well catered for by experts. We think that for some time to come the operation will remain almost entirely in the hands of the latter, and that less space might be devoted to it in any work not intended for specialists.

The drawback of composite authorship in a medical work written for one class of the profession is that each writer is apt to devote himself to his section, while forgetting his reader. This book has been so long and favourably known that we venture to hope that in the next edition Dr. Macnaughton-Jones will insist more strongly on his editorial prerogatives, or else give us the entire work from his own pen—a task for which his thirty-four years' constant work in otology, and his evergreen interest and activity in it, render him eminently suitable.

Atlas and Epitome of Diseases of the Mouth, Pharynx, and Nose. By Dr. L. GRÜNWALD, of Munich. Second edition, revised and enlarged. Edited by JAMES E. NEWCOMB, M.D., Cornell University Medical College. With 102 illustrations on 42 lithographic plates, and 41 figures in the text. London: W. B. Saunders and Co. 1903.

THE profession has for some time been supplied with many excellent atlases of diseases of the larynx. Recently, in these pages, we gave particular praise to the most recent one, by the author of the work we are now considering. In turning his attention to the pictorial representation of diseases of the nose Dr. Grünwald has followed Gerber and Krieg in a courageous attempt to depict what might be considered the impossible. The view obtained, even by the most expert, of the interior of a nasal fossa is necessarily a composite one; and, unless we have a succession of pictures to represent one condition, it is physically impossible to represent in a drawing what the eye successively takes in during its scrutiny of this narrow, tortuous funnel. Now, to the rhinologist these pictures are superfluous, and to the student they are less helpful than some good, semi-diagrammatic outlines, which would not attempt to represent the exact appearances, but would train him in seeing them. However, having relieved our minds of this pious opinion in reference to the drawings of nasal lesions, we can give unstinted praise to most of the representations of the mouth, pharynx, and post-nasal space. In the epitome which follows Dr. Grünwald is to be complimented on his courage in departing from the beaten track in many instances, and on originality of view, based on observation and thought. Indeed, it might possibly be thought that for an epitome of the subject there is too much pathology, and too little of symptoms, diagnosis, and treatment. But this is a fault in the right direction: the work is educative; it is distinctly stimulating; and it is not intended to replace text-books on the subject.

There is little for us to criticise in detail, but we think it unwise to advise that the maxillary antrum should be laid open, if syringing through a tooth-socket has failed to arrest suppuration "after two or three weeks" (page 102). We know of cases which have required much longer, and in which perseverance in this simple measure has resulted in a lasting cure. It is also surprising to read that suppuration in the frontal sinus is "extremely rare" (page 111). In London clinics it certainly is not as common as antral suppuration, but it is of such everyday occurrence that it certainly cannot be termed rare; post-mortem examinations also support this view. On page 176 there is mention of the "predilection of cancer for the third and fourth decades." Surely this should have been the fifth and sixth decades. There is a typographical error on page 25, where the "exploratory pressure of the lungs" is mentioned, when, of course, it is intended to refer to "expiratory."

The translator has done his work satisfactorily, and the English edition reads easily. We are not sure that his intercalated remarks are always illuminating. For instance, he only confuses the reader by adding after "¼ liter" the word "quart" in brackets. This might mean that a ¼ liter is equal to a quart, instead of being a ¼ of a quart. How often must we point out to rhinological writers that the plural of "meatus" is not "meati" (page 95). If they wish to stick to their Latin and be correct, they should write *meatūs*; but they would be wiser to Anglicise the word and make the plural of it "meatuses." We cannot agree with the translator's note on page 142 that the symptoms of tonsillar and peritonsillar abscesses "are practically the same." In the description of Plate 29 we must point out that "jaw" is rather a loose term to employ, and does not distinctly indicate the superior maxilla. To say of a set of tongue-depressors that it "comes in three sizes" (page 34) is rendering the original German very literally; and to talk of Kuhnt's method as "obliteration of the cavity after Kuhnt" (page 116) is too palpably a literal translation of an idiomatic German phrase. But otherwise the translation has been made judiciously. Even those, however, who welcome the American method of spelling may find such words as "mustache" (page 29) and "licorice" (page 140) appear new to them.

In conclusion, many of the illustrations are excellent, and will be helpful to those desirous of perfecting themselves in the subject; while the letterpress will show anyone the true scientific spirit in which to approach diseases of the upper air-passages. We can, therefore, cordially commend the book to our readers.

Obstinate Hicccough: the Physiology, Pathology, and Treatment. By L. F. B. Knuthson, M.D. Edin. Pp. 169. J. and A. Churchill. 1902. Price 6s.

This is a laborious collection of one hundred and fifty cases of hiccough, culled from various sources, and prefaced by a case of the author's which it is calculated hiccoughed no less than 480,000 times in the month preceding death. Possibly the collection of cases and the list of remedies thought to have been successful may be of use to someone. The subject is one which previously seemed to be interesting, but this volume is admirably calculated to correct such a view.

Operative Surgery. By HERBERT WILLIAM ALLINGHAM, F.R.C.S., Surgeon to the Household of His Majesty the King, Surgeon in Ordinary to His Royal Highness the Prince of Wales, Senior Assistant Surgeon and Lecturer on Operative Surgery, St. George's Hospital. Demy 8vo. Pp. 367. Illustrations 215. London: Baillière, Tindall, and Cox. Price 7s. 6d. net.

THIS little work, of convenient size and usefully bound, is intended to be of service "to surgeons wishing to hastily look up" (the split infinitive is the author's) the leading features of an operation." No lengthy descriptions are given, and no attempt is made to include all the operations practised. The plan followed in each instance is as follows:—The anatomy of the region is succinctly given in small print; a list of the operations follows; each operation is adequately described and "memoranda" are added mentioning the difficulties and giving the special hints which are likely to be helpful. The great majority of the descriptions are illustrated, the drawings, which are in most cases clear and informing, being the work of Mr. Lockhart Mummery, F.R.C.S. Though there is much to be said in favour of this book, it is doubtful whether it will displace the text-books already in vogue. For in more instances than one brevity becomes curtness; there is a sacrifice of essentials, some descriptions lack clearness, and there are omissions of operations of the first importance. For example, there is no mention of Wyeth's pins for transfixion in amputation at the hip, nor of Lockwood's operation for femoral hernia, an operation which by many surgeons is considered the only one of real value. The method described for the performance of jejunostomy is entirely obsolete, while in the memoranda following the description it is said that there is risk of leakage if the opening is larger than a No. 8 catheter. The descriptions and the illustrations of the operation for hare-lip are trivial; there is no mention of the "pout" which is so necessary to ultimate success. The splitting of muscles in inguinal colotomy, after the manner of McBurney, is not mentioned. Krönlien is spelt Kronlein, and on pages 112 and 113 a late distinguished President of the Royal College of Surgeons is called "Skeg." We have mentioned the defects rather than the virtues of this work. The latter, it is true, are not inconsiderable, but in a text-book of this kind error should not be allowed to creep in. If a second edition is called for, we should expect that a careful revision would result in the production of a really helpful little work.

International Clinics. Vol. III. 12th series. London: J. B. Lippincott & Co. 1902. Price 10s. 6d.

THE present volume justifies its claim to be "international," for it contains among many American names those of Dr. Borissof of St. Petersburg, Professor Cardarelli of Naples, Drs. Debove, Dieulafoi, and Hallopeau of Paris, and others. The papers are of the usual standard of interest. We may mention especially one by Professor Dieulafoi on the diagnosis of tubercular pleurisy. He not only alludes to the tuberculin test as being of use, but also describes the so-called "cyto-diagnostic" method. It is found that in a pleural effusion, due to passive exudation, there are contained many cast-off cells from the wall of the cavity; in an exudation due to infective agents, such as the streptococci, typhoid-bacilli, &c., there is a majority of multi-nuclear cells present; while in a tubercular effusion the greater number of the leucocytes present are of the kind known as lymphocytes. This method of diagnosis is of considerable clinical importance. Other interesting papers are those by Dr. J. J. Walsh on "Insect Pests of Human Beings," and by J. H. Brinton on "The Effects (on the skin) of Firearms at Short Range."

Essentials of Human Physiology for Medical Students. By D. NOËL PATON, M.D., B.Sc., F.R.C.P. Edin. Pp. 376. 110 Illustrations. Edinburgh: William Green and Sons. 1903.

IT is now recognised by all the younger teachers of physiology that the scope of physiology has become so wide that the medical student must, to a certain extent, "limit his attention to matters which have a direct bearing upon his professional work." The educational value of physiology will not be diminished, even if less stress be laid upon the physiology of muscle and nerve; respiration, secretion, metabolism, reproduction, and nervous action are subjects of the utmost importance to the medical man, and supply problems which will exercise and develop the best faculties of the mind. To these views Dr. Noël Paton has endeavoured to give effect, and at the same time he has attempted "to bring the practical and systematic study into closer relationship by constant references to the practical work which the student must undertake." In our opinion the value of the book in the latter respect would have been increased if classical experiments and more important physiological apparatus had been described and figured. As it is, there is a lack of sequence, which is not filled by the abrupt reference "(Practical Exercise)." The ordinary student will not during his reading carry out this instruction and refer to another book for an account of the practical exercise.

The "essentials" are divided into three parts; the first sections deal with Protoplasm, the Cell, the Tissues, the Special Senses, and the Central Nervous System; the second part, the Nutrition of the Tissues, is concerned with the Blood and Lymph, the Circulation, Respiration, Food, its digestion and utilisation, and excretion; the third part is on Reproduction. This subdivision is one that can be defended, but it is doubtful whether the student will find it easy to study the Special Senses and the Central Nervous System before the so-called vegetative processes. It is an excellent feature of the book that so much space is given to the subject of Food, Digestion, Metabolism, and Dietetics. The practical importance of these subjects is obvious, and a sound foundation for their study and application to medicine must be laid by the physiologist. Only four pages, however, are given to the third part, Reproduction. Here, in our opinion, the author fails to carry out his aim. Is not reproduction of "cardinal importance in medicine and surgery"? Is it not one of the "matters which have a direct bearing upon professional work"? No doubt the answer would be that this subject falls within the province of the teacher of obstetrics, who, practising both medicine and surgery, is well qualified to point out its bearing on those subjects. This modern view is widespread; we are convinced, however, that the physiologists of the earlier part of the nineteenth century were right in considering the subject of reproduction as an essential part of a course of lectures on physiology. The neglect of this subject in modern physiological text-books is to be deplored. In the eyes of many candidates, and even of some examiners, fœtal respiration, circulation, and nutrition are questions of midwifery. This severance of reproduction from a science as generous as physiology, and its inclusion in a somewhat narrow speciality, have resulted in a very limited view of most important phenomena. Reproduction may, from a biological point of view, be stated to be the sole aim, the climax, of living processes ; it must be considered in any logical system of physiology.

The style of writing in the book before us is clear and concise ; it may be somewhat dogmatic, but this is generally allowed to be an advantage in an elementary text-book. There are a few misprints and mistakes; the head-line "central nerve," which is found on a dozen or more pages, is, no doubt, a printer's error for "central nervous system," and the mistake in the formula for palmation is probably of similar origin. The printing, paper, and binding are excellent. We feel sure that the book will be popular with many medical students.

Ocular Therapeutics according to the most Recent Discoveries. By Dr. A. DARIER. Translated by SYDNEY STEPHENSON, M.B., C.M. London : J. and A. Churchill. 1903. Price 10s. 6d. nett.

THIS book of 272 pages is a more or less literal translation made from the second French edition of Dr. Darier's "*Leçons de Thérapeutique Oculaire.*" The translator is Mr. Sydney Stephenson. It consists of 26 lectures and is arranged thus, while at the head of each chapter is a summary of the lecture showing at a glance the ground covered by each. The work has been awarded the Deportes Prize, 1902, by the French Academy of Medicine as "the best work published on practical therapeutics." Dr. Darier is such a well-known exponent of ocular therapeutics in France as well as in England, and Mr. Stephenson is also so well known in this connection, that we confidently expect the book to be thoroughly up to date. Such indeed it is, and it contains a vast amount of useful information derived from practical experience. The drugs that have been advocated of recent years and that have proved themselves of more or less use in ophthalmic work, are so numerous that there can be but few practitioners who have given even the majority of them a fair trial. Dr. Darier has certainly done more than most to find out which are useless and which are of value, and it is a great help to read his conclusions. No pains have been spared in acquiring the knowledge, and we find the actions of all sorts of drugs which are by no means in daily use carefully discussed.

Dr. Darier is a most enthusiastic supporter of subconjunctival injections, and the technique of this procedure is minutely described. He is also a very strong advocate of hypodermic and intravenous injections of certain drugs, notably of mercury, which he finds most convenient to use in the form of the cyanide, as this is far less irritating than the sublimate. He finds that he is well able to control the pain of the subconjunctival injections by the use of a few drops of acoïne added to each dose.

He has practically given up the use of nitrate of silver, substituting for it the numerous other silver-preparations, of which he holds protargol to be the most valuable. Massage he finds most useful in many cases, and it is of particular benefit in reducing intra-ocular tension in cases of glaucoma. The English reader may find it a little confusing to have all the proportions given in the metric system, as is done throughout. If, however, authors would adopt this most rational system universally, we should soon find that everyone would be as familiar with the weights and measures as most people are now with the centigrade scale, and we should cease to hear complaints on this score.

The book requires to be read to appreciate the profound belief which the author has in the numerous drugs that he advocates; and if they give as good results in others' hands as in his, it seems that nearly every pathological condition will yield to their influence. Whether universal experience will prove such to be the case remains to be seen. For the work, as a whole, we have nothing but praise: it is interesting, complete, and well translated. There is no other book in English to equal it; and we heartily congratulate Mr. Stephenson on the edition he has brought out, which supplies at once an excellent book and a real want.

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THE PRACTITIONER.

DECEMBER, 1903.

A LECTURE ON THE MENTAL DISORDERS ASSOCIATED WITH DECAY.[1]

By G. H. SAVAGE, M.D., F.R C.P.,

Consulting Physician to Guy's Hospital, and to the Asylum for Idiots, Earlswood.

GENTLEMEN,

TO-DAY I have to consider the mental disorders associated with bodily and mental decay. Of course, the most general and common example is met with in senile decay; but when one uses the term "senile," one has to remember that this, after all, is relative; that whereas one man may be worn out at forty, another may be young at sixty or seventy. Again, one has to remember that mental decay depends upon the original constitution to a great extent. There are some individuals who seem to be built up along such lines that they break down first of all on their nervous side. One recognises that one man dies by his brain, another dies by his body. And I would point out that in families you get very clear evidence of this; there are some families whose members die out along their nervous lines. I have known one or two rather distinguished families, the members of which lived to extreme old age; but they invariably developed the melancholic symptoms which precede true dementia.

Premature senility produces exactly the same symptoms to which ordinary senility gives rise. The mental disorder of premature senility may be induced by worry, but much more commonly it is dependent upon alcohol, and very commonly upon syphilis. I have sometimes quoted the Shakesperian lines that "first we ripe and ripe, and then we rot and rot," and pointed out that syphilis and alcohol seem to have the power of ripening to rottenness the nervous system above any other part. Therefore you may get any one of the symptoms of mental decay at almost any time; the insanities of decay and old age occurring at any age—I may say from thirty to ninety. The time of their appearance depends not only upon the hereditary constitution of the individual, but it may and does depend upon surroundings. I am in the habit of pointing out to students at hospitals that the decay of a house depends very much upon its environment. For example, a house built in an open plain may last for long, and perhaps some of its apparently most fragile parts will outlast those seemingly far stronger. That is to say, the windows may survive the doors, and the doors may even survive the roof. But let that house be in the neighbourhood of a city, then the windows go first. And so it is with some cases tending to the mental breakdown of age. If such people can live in the placid calm of a country place, they may go on almost indefinitely, and only very slowly become senile. But let the same sort of persons be in the rush of the city and the case will be different. I will take as an example a family in which were two brothers. One of them lives the quiet life of a country squire; and at eighty he begins to be querulous, restless, forgetful. He becomes more fretful, more extravagant in his actions, and finally becomes childish, but only very slowly. All this takes some years to develop. The other has been full of life, of interest, and of work; and he comes to you about the same age, but used up by the thousand and one things which have occupied his attention. He, on the other hand, degenerates rapidly; he becomes childish in six or eight months, whereas the brother has taken as many years to break down.

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[1] Delivered at the Medical Graduates' College and Polyclinic.

Of course, it is generally considered that loss of memory is about the most characteristic defect associated with senility. Undoubtedly that is true, and it is a very important thing to remember. But decay of memory is not always in direct relationship, if one may use the term, to decay of brain. One of the points of characteristic difference between general paralysis of the insane and senile decay is, that in senile decay the memory fails so much more markedly and completely than it does in general paralysis of the insane. Very commonly I see cases of advanced general paralysis with comparatively slight defect of recent memory; whereas, on the other hand, the most characteristic symptom of senile loss of memory is the defect of the memory for recent events. It is an important thing to remember, this progressive defect of memory; and I am inclined to think that there are two or three other points that are noteworthy, not only from their symptomatic interest, but also from their medico-legal importance. There is apparently a "denudation," as the geologists say. You may have a skeleton in the cupboard that the sands of time have covered up; but, to carry out the simile, the waves of denudation once more disclose the skeleton. A man's wife was untrue to him forty years ago. It was forgiven, apparently forgotten. The family has grown up, and the old man, when seventy years of age, begins to accuse his wife of recent infidelity, accusing her of sin with the very man with whom she was guilty forty years before. As time goes on, he forgets; the skeleton is washed away, and peace comes once more. So one has seen in a similar way a man who had a prodigal daughter, who married a man of whom he did not approve. He would have nothing to do with her, and she went with her husband to the colonies. When middle-aged, her children and her husband having died, she returned to her father, like the prodigal son. He forgave her, and she attended upon him in his weakness and his illness, and he again became devotedly attached to her, as in truth he ever had been. Then age came on and left him with a decreasing memory. He now forgets the good deeds of the immediate present, and only thinks of her as a prodigal daughter, and will have none of her. Her mere approach causes excitement and worry, so that in the end she has to go away. This man, if he had been able to do what he wished, would have alienated his property from his child, remembering only the distant past and not the more recent events. Hence the vital importance, from a medico-legal point of view, of recognising these and similar defects of memory.

Another point of importance, when considering the vagaries of memory among old men, is that you may get an accentuation of the old memories, so that they are brought out more clearly. Take an example:—I remember a man who was an officer in the Crimean war, who came to be garrulous and forgetful of ordinary things; but he had a most vivid memory of the events in the trenches, even to the losing of a button in a certain engagement. Many similarly-trivial incidents were brought out by him. And when one asked his family, "Is this a mere repetition of what he has been telling you during the last thirty years?" they replied, "No." Of course one could not verify many of the things which he said, because his associates had passed away, but I got some of them verified, and there was no doubt of the truth of them. There seemed to be an accentuation, or you might say improvement, of memory for the old things, and decay in memory for the present.

Here one may observe that senile decay and the mental disorder associated with it are accentuated by alcoholism. In fact alcoholic dementia is in many respects absolutely parallel in its symptoms to senile dementia. Take an example:—In senile dementia it is common to meet with a patient "babbling of green fields" and talking of the past; he tells you he has been for a long walk, that he has done this and that; that he has been to his office this morning, and interviewed this person and that; that he has transacted business; and yet all the time he has not left his chair for weeks. I saw last week such a case in an old man who had not been out of his house for five or six months. I asked him, "When were you at your office last?" "I should have been there now, if you had not been coming. Of course I was there yesterday." "Where else have you been recently?" "I dined out last night; I had to go to my cousin's"—living at so-and-so, twenty miles away. This is a very common phase of senile dementia, and it is even more clearly defined in alcoholic dementia, *i.e.*, the so-called "romantic" lying, and also the imaginative mental weakness met with in both these cases.

I now come to a most important point, from a medico-legal point of view, the extent to which the loss of memory may affect property. Some little time ago I had to see a man who was suffering from senile dementia. The question was whether he was, or was not, fit to administer his affairs. I informed myself as to the number of children he had. He had had five; only two of them were living. I said to him, "How many children have you?" He said, "Five." "How shall you distribute your property?" "I shall distribute it evenly." "John, is he alive?" "Certainly." "And Jane?" "Yes."

Both of them had in reality been dead for years. It is an equally common thing for women to make mistakes in regard to their relations. I do not know why it is, but, as I have pointed out elsewhere, this mistaking of identity is in fact strangely common among women, perhaps more so on the whole than among men. A case of puerperal insanity will often claim the doctor for her husband: such cases in fact constantly mistake identities of all sorts. Similarly women in old age frequently say, "You are my brother," or "You are my uncle." One cannot but have known cases in which advantage has been taken of such recognition. We will not suppose it to be a doctor; but to any person in close relationship with some such senile patient, the patient might say one day:—"You are my dear, dear uncle; I shall leave you all my property." One cannot be sure that advantage will not be taken of such a condition of things; and, therefore, it is important to study closely the medico-legal aspect of such defects of memory.

Another point is: Under what conditions can you treat such patients? This, of course, is the practical question. Nearly everybody seems to think that persons have the right to keep an old man or woman, who is demented, without certification. But certainly that is not true. Possibly the time may come when registration, as to where a senile dement is, will be found sufficient; but at present no one has any right to keep a senile dement in his house for any pay or reward. If he does, he is breaking the law.

Up to the present I have given, as it were, a general introduction to the subject of mental decay, as depending upon senility. The term "dissolution" of course is one that is habitually used in relationship to these cases; or, as some say, the process may be looked upon as mental reduction from a higher to a lower level. The most common symptom, probably, in many cases is loss of higher self-control, so that there may be a kind of inversion of such symptoms as I referred to previously, when I spoke of the disorders associated with development—hysterical conditions, moral perversions—and one gets exactly the same sort of thing in senile cases. Senile hysteria is not at all uncommon. It must be remembered, however, that, the connecting links are very simple between what may be called the normal and the exaggerated normal, which becomes disorder. I recently saw an old gentleman (about 80 years of age) who, in this kind of way, laughs and smiles about very little things. I should think there are very few weeks in my life when I do not see men and women who are distinctly hysterical upon very slight provocation. Unfortunately, by this loss of self-control, uncontrollable passions are sometimes liberated. When one is asked to see a hysterical woman, one is often told that her temper is devilish. At a particular time she may be amiable; but without the slightest reason she becomes violent and destructive, and smashes things, and for a short time is really maniacal. Or one is asked: "What shall I do with my father? He has knocked the butler down and he has smashed valuable glass, and he sees it was wrong, but there is no guarantee that he will not do it again to-morrow." Thus, from a medico-legal point of view, we come to the important point that a man may commit an assault and battery, or severe aggravated assault, or even homicide, because he has lost his self-control owing to increasing age.

Quite common, indeed, among well-marked senile cases is a more or less typical mania. And you often get quite a similar type of mania to what you meet with in the young. There is, for example, a hysterical emotional mania, in which there is found a noisy boisterous destructiveness, with disregard of all the finer feelings. Amongst both young and old will you find bad language and bad habits, dirtiness, masturbation, and obscene language, with violent conduct, including exposure of the person. Therefore it becomes sometimes absolutely necessary to send the old cases to asylums, simply and solely because of the excitement. What indeed are you to do? Of course I would put it down as an axiom that I would never send a patient over 70 or 75 to an asylum, if he has money enough to keep him at home with nurses. It seems hard lines, if a man has lived 70 or 80 years out of an asylum, that he should die in one after all. But there again it depends considerably upon the length of the purse. Some cases of senile mania die off quickly. If there is a rapid running pulse and a refusal to take food and a dry tongue, you may be pretty certain that such a man need not be sent to an asylum, for he has only a week or so to live. Symptoms of this type may be 'ushered in by acute mania; or they may follow a feverish attack, such as influenza, or an apoplectic seizure, or a local cerebral softening. Again, one has seen a case of this sort develop an ephemeral mania, which passed off in a day or two; and one could not work out the causes. Then came another apoplectic seizure and death. There was a small hæmorrhage over the surface at first, which was absorbed, and then another hæmorrhage came on which killed the patient. Thus you find both maniacal attacks which depend simply upon senile decay, and senile attacks of mania following some hæmorrhage or local softening. The prognosis, of course, will vary directly with the amount or gravity of the local lesion of the brain.

A certain number of these old men who have mania pass —I was going to say, unfortunately pass—into a condition of chronic mania, of a mild type possibly, but of such a distressing form to the relations that they almost invariably have to be sent away. For example, a man who has been a quiet, restful fellow, who has been full of interest in life, comes to somewhere about eighty years of age. He has a sharp attack of mania, and after the acuter symptoms pass off he remains a restless, almost mindless, human being, who rushes upstairs, rushes downstairs; wants to get out, but no sooner is out than he comes back again and takes off his clothes. There is not a moment's rest where he is. He eats and drinks well, and sleeps well, while he is asleep; but that is only for a short time, for he seems in five or six hours to be able to get as much rest as he wants, and then he goes on again for another nineteen hours or so, without aim or object, restlessly moving about. Certainly these cases are common enough, and where you find cases of restless old people, you may say that probably they will remain restless to the end.

You might perhaps expect, in consequence of acute mania passing into chronic mania, to find established a certain calm of dementia, partially dependent upon the nerve-exhaustion; but that does not happen in many of these cases. They will go on for a long time, until some accident kills them. Such patients remind one of one's dream-sensations. Some of you have probably read Horace Hutchinson, of golfing fame, on *Dreams and their Meaning*. He gives the many common dream-experiences, such as I meet with in my friends.

Personally I find it quite a common dream to go on finding that you are continually doing some trivial thing and not satisfying yourself over it. An example is packing a portmanteau to go by train. You pack it, and then take the things out, and then you pack it again ; and, though you go on packing, you never complete it. These old people, too, go on in that dreamlike way still; they go on, but they never finish. Unfortunately many of these cases with loss of control and mania become, as I have told you, regardless of decency, regardless of cleanliness at first ; they urinate or defæcate anywhere, when they feel inclined. And then, still worse, they may be passively, or they may be actively, obscene. I own that for years I have declined to defend cases of this kind in the police-courts—I am afraid from rather a cowardly point of view, but I do not like them. Generally people will talk and say, "Oh ! yes, if the prisoner was not a rich man who could get an expert witness, he would be sent to jail." I own it is rather cowardly not to defend them, but I prefer that younger men should do it now. Certainly, it is very sad to see a man who has led a perfectly reputable life, until he has come to seventy or eighty years of age, punished because he exposes himself indecently in front of a window, there being a milliner's shop opposite. You know these cases are irresponsible, as shown by such an instance as the following :—An old man about whom I was consulted a good many years ago indecently exposed himself in Kensington Gardens, or somewhere in the west of London, and it was noticed that he did it repeatedly. It was thought that he did it specially before women and nursemaids. If the desire struck him to urinate, he would urinate anywhere. But it was contended that all this was deliberate, indecent exposure. The case was brought up before the magistrate, and the man was remanded and bail was accepted. He was allowed to go home by himself; and between leaving the jail and reaching home he did it again. So you may take it for granted that either his memory or his impressionability was so defective that he could hardly be considered responsible. It is rather startling to have to recognise that our reputations may depend so much upon our arteries. Take an example :—An old clergyman, who lived a quiet peaceful life, degenerated slowly. He was able to read the lessons, and his chief inhibitor was a good wife. That inhibition was removed by her death, and then he began to do foolish things. Some time afterwards he wandered away, and did not attend his church at all; and his curates never knew where he was. Then he fell into the hands of the police, and was said to have been drunk at the Alhambra. There is no doubt he had got into all sorts of bad company who robbed him and gave him drink. And yet that man had been as worthy and good a fellow as anyone could wish to meet with, until his arteries began to go. Later on, he had an apoplectic stroke which, fortunately, ended the matter. "Thou shalt not" is a stimulus to action always, and the life of a clergyman in a country place like that may be so hedged round by common demands that the loss of higher control is exhibited by a very definite swing of the pendulum in the other direction.

One has to remember that there is a great difficulty sometimes in making a diagnosis between senile mania and senile general paralysis of the insane. I would say that there is a special disease, senile general paralysis of the insane, which is very difficult to distinguish from mania. In senile mania you may, and not infrequently do, get great exaltation of ideas, a great amount of boastfulness. Let me tell you of an example which I thought once was a case of general paralysis. A man of something under seventy years of age had been in a public office before the time when I saw him. He was a tall man, and he said to me "I am very tall; I am the tallest man in England. Would you like me to make you tall?" And he began waving his arms about. He said "I would do anything for you." He had grand ideas: he was benevolent in the extreme, but he was also rather licentious and lascivious, and obscene in his actions and talk. There was some tremor of the tongue and lips, I thought, but there was no inequality of pupils and no history of syphilis, though there was a history of previous attacks. Therefore after more mature consideration I concluded that it was a case of senile mania and not one of senile general paralysis. But one sees cases of unmistakable senile general paralysis, in which the syphilitic lesion seems to have lain dormant 30 or 40 years before exhibiting itself in degeneration of the arteries of a syphilitic type, leading to general paralysis and death.

Again, some of the most difficult cases are those of senile epilepsy. Just as you may get senile softening of the brain, giving rise to some monoplegia or hemiplegia, so you may get local softening or some local change in a cortical area which will give rise to very definite epileptic seizures. Probably it has come under the experience of many of you to have seen cases of old men who have had attacks of hemiplegia. They seem to be getting on well for some months; then they have a series of truly epileptic fits, and begin rapidly to go to the bad. One of my old friends, a leading London doctor, had an apoplectic seizure, from which he seemed to recover. A year went on, and then he got epileptiform seizures of which he knew the meaning. He recovered for a time. Fresh seizures came on, and he died. There may be considerable difficulty in differentiating such cases as these from what might be called the general paralysis of old age. For it is to be remembered that you may have general paralysis occurring in old patients, though it is not common.

Again, probably one of the most common forms of mental disorder associated with senile decay is melancholia in one or other of its forms. With adolescence it is usual to meet with patients who have various forms of hypochondriasis. Sexual hypochondriasis is quite common among young men, who believe themselves to be impotent, or suffering from some venereal or other disease.

And here I may say that it is interesting to note that one of the not-uncommon symptoms of progressive senile decay is hallucinations. Hallucinations may be hallucinations of sight, or of hearing, or the like. Hallucinations of smell are not very uncommon, and such hallucinations of smell in many cases of progressive senile decay are associated with hypochondriacal ideas that everybody else perceives the bad odour; that they themselves have got venereal disease; that they are foci of syphilis, and that people will come and tear them to pieces because they are spreading syphilis broadcast amongst the population. Others will seclude themselves altogether, and become senile solitaries. There may in fact be almost any form of melancholia with old age. Oddly enough, the exaggerated hypochondriacal symptom which is most common is the idea of occlusion or obstruction of the bowels. Whether that is as common in other countries as it is in England I do not know; but one always thinks of Sir Samuel Wilks's saying that the Englishman seemed to be brought up with the idea that there was a rotation of the earth in twenty-four hours, and that there should be a rotation of the bowels in the same time, and that one was as essential as the other. At any rate one knows that these ideas of obstruction do exist. Patients refuse their food; and that may be one of the reasons why a patient suffering from senile mental disorder should be sent to an asylum—it may, indeed, be simply and solely because he refuses his food obstinately, and has to be fed artificially. If you find great difficulty of that kind, it is almost certain that you will have to send the person to an asylum, however old he may be.

This refusal to take food may come from one of two definite reasons—either because he thinks the food stinks—that is to say, he has hallucinations of smell—or because he thinks his bowels are obstructed and that nothing passes through him. Of course it is a disorder of feeling, such as I shall have occasion to refer to later. I remember as a young man at Bethlem saying that I would try to follow Solomon and answer a fool according to his folly. In one or two cases where patients said nothing passed through their bowels I said, "Very well! There is a room and there is a bed-pan, and you are going to sit there for the next week." Well, something was passed. I thought I had won my point. But the patient said "No, somebody else did that; I am not responsible for it." I did not convince him; he felt clearly he had an obstruction, and that was an end of it as far as he was concerned. I have known a man commit suicide by ripping himself open, simply because he was so sure that he was obstructed, and that this act of his was the only way out of it. One has seen cases in which the condition has alternated or recurred; in which an old man, instead of having an attack of gout, got an attack of hypochondriasis, with an idea that his bowels were stopped, or that his throat was stopped. I remember one old man who came to Bethlem and obtained his own certificate, saying that he was sure that he would never be able to swallow any more. He came on two occasions to get certificates to admit him into Bethlem for his terminal treatment, as he called it.

The most common form that melancholia takes in the old is a dread of ruin, poverty, the workhouse. Nothing is more absolutely characteristic, I was going to say, of senile melancholia than the idea of the workhouse. But there may be no pretext whatever for it. Such a man is told, "You have a good house." "No," he will say, "I am ruined." Here again I would suggest that no good, or very little good, follows from arguing with the patient. There was a man whom I had to see in Surrey some years ago. When I arrived he said, "I am sorry they have brought you down, because you will not get any fee. There is no money. Did you see the bailiffs at the gate?" "No." "Well, they are there." "But what about your beautiful house," said I, "and did not your carriage meet me at the station?" "Ah, but it is not my carriage any more; it is in the hands of friends." "Come now! do you really mean to say you believe yourself to be ruined?" "Why certainly," said he, "I *know* I am." I suggested that I should see him again in a week, and if in the meantime I got a statement from his bankers as to his funds would he then admit that he had been mistaken. "Well," he said, "I shall not see you next week when you come, but you will find that I am right." And so I went away. and came again afterwards having provided myself with a banker's book and also his manager's report. That report was the very best the firm had ever had in its existence. There was a current account in that man's favour of £10,500. "Well," said I, "What do you think of that?" "Ah," said he sadly, "that was *yesterday*." This patient got well afterwards, at all events temporarily. And I remember him talking somewhat after this fashion. "You know it is all very well to argue, but if you feel you are ruined, and that you have got nothing, I do not see that arguing applies at all." Such a patient's feeling of being without money is as definite as the feeling of the man who gets nothing through his bowels.

But occasionally we have to discount some of these delusions as being partial facts. One is again reminded of Sir Samuel Wilks, who tells of a case where he was called down to see an old man who said he was ruined, and that his carriage was not his, and that he was fraudulent. Like myself, Wilks said: "Look here, at your beautiful house and beautiful grounds, and everything sumptuous." This patient was actually ill, and was not sent to the asylum; and he was also actually fraudulent, and had lived on trust-money until he had come to the end of it. Obviously, one has to be sure of one's facts. The patient had an exaggerated interpretation of what was absolutely true. And, in a similar way, you may find men accusing themselves of crime. A man may say, as one did to me recently: "I am an altogether wicked and abandoned man." When he was a young man, he had had a mistress; but this was forgotten and forgiven, and the fact was known and recognised by his wife when he married her. Now, however, with the onset of senile decay, appears the skeleton in the cupboard, and he says he is an abandoned person, and that he has been professing Christianity, and at the same time he has been chairman of this and that society. And one meets with people who seem to be always looking into their lives for a peg upon which to hang the interpretation of their miseries.

In relation to these cases of melancholia, plenty of food should be given, and bed and stimulants, but not stimulating surroundings. I am horrified to see old men who are suffering from early senile melancholia sent to enjoy themselves in Paris or Rome. They are much better in bed, and they are very much better looked after, as a rule, by female than by male attendants. I say that in looking after a senile melancholic or dement you can hardly go too far in the matter of careful nursing. I frequently say: "Let the nurses have long strings; let the old man feel that he is being treated." The patient should be put to bed and fed carefully. Another thing which is very important is, that such patients should have quite a liberal amount of alcohol. I have known alcohol do more in the way of giving rest and sleep at night for some of these old patients than anything else. A certain number of these patients die. Just as I mentioned that you may find maniacal patients wearing themselves out, so a melancholic patient may not infrequently wear himself out. But bed, nursing, restful surroundings, and warmth are practically all you can do.

You will remember that, as with mania you may get established or chronic mania, so also you may get chronic melancholia. I remember a man who died nearer 90 than 80 years of age, at Bethlem, and who for some years had been one of the incurable patients. He had begun with the fear that there would be no food for him, and gradually his care in the matter of food became a sort of clockwork business. When called in the morning the first thing he would say was: "Breakfast at 8 o'clock, breakfast at 8 o'clock, breakfast at 8 o'clock." And when it got within five minutes of 8 o'clock he would shout at the top of his voice: "Breakfast at 8 o'clock!" Then after breakfast he would start repeating: "Beef-tea at 11 o'clock"; and so on through the day. And one has known similar cases of chronic melancholia in which you hardly notice the melancholia. Just as one has heard of people describing a condition as fever in which there was no fever present, so you may have melancholia without melancholy. The visible sign is often a monotonous habit of repeating things. I remember another patient who was continually saying: "I do not know what to do, I do not know what to do." But she ate, drank, and slept well. She passed into a state of weak-mindedness, with a melancholic expression. And it is well to remember that many of these patients who pass into senile melancholia of a chronic type do not suffer. The patient's friends will say: "Poor thing!" But these cases do not suffer any more than a clock suffers from ticking.

A considerable number of patients pass into a state of mental weakness after apoplexy. Some have doubted whether any man is quite the same intellectually after an apoplectic seizure, whether it be from hæmorrhage or from softening. I think it is rare for a man to be quite on the same plane intellectually afterwards as he was before, but it is not impossible. After an apoplectic seizure you may find almost any form of mental disorder. There may be acute mania, but it is much more common to find progressive mental weakness, with very marked irritability of temper, loss of memory, loss of motor powers, and loss of self-control, as the chief features. But there may be in fact almost any form of mental disorder.

There are just one or two other things upon which I want to enlarge somewhat before we leave. One word as to the disorders associated with the sensory organs—hallucinations, in fact. It is not uncommon for me to see patients who are troubled by visual hallucinations. Here again is an illustration of a point which I mentioned previously, for in young men, as I have told you, visual hallucinations are common. Auditory hallucinations are the most common in ordinary insanity, but in the insanity of youth there are often feverish states, and with these and similar conditions of high temperature you get visual hallucinations. In alcoholic cases, also, you are aware that hallucinations of sight are common. In old age you will find hallucinations of sight very frequently. Five years ago I was called a long distance to see a very wealthy old man—his will was afterwards proved for a very large amount. He had passed through the stage of restlessness; he was fidgety, and was hanging and re-hanging his pictures constantly, worrying the gardeners and all the other people about the house. Then he became suspicious that people were watching him. He therefore got a revolver with which to shoot burglars. The people wanted to send him to an asylum. I prevented his going to an asylum, but as he was the owner of such a large property, and as his memory was defective and he was weak-minded, I concluded that it would be wrong for him to have the administration of such a large estate. Well, I went to see him. He received me; I cannot say he welcomed me. When I told him that I was a doctor, he told me that he had seen a good many of that sort, and that he did not want to see any more, because doctors tried to make out that he was mad, and he was not. I told him I had come to make out that he was not mad. After a little time we became friendly. I said "You were rather troubled about things in your garden?" "Yes," he said; "do you see those rabbits?" "No." "Do you not see that old doe with a lot of little ones running into the laurels?" I said, "There are none, but shall I get a gun?" He said, "No, I see them, but they are not there." Then he asked me if I saw "that cistern up there." I said, "Yes, I have been told about that." "Well, you can see water running across there?" "No," I said. "But I think there is water. Yet I tried some blotting-paper and it is not wet now." I did not think this man was dangerous; I thought him harmless, and I did not think that because he was always trying to correct certain sensory impressions he should be sent to an asylum. He was not sent in fact, and lived a free man till his death, and I think that his property was justly distributed. The same week I saw a lawyer, who said, "I know what you have come to see me about. I know you are a mental doctor, and my symptoms are mental, and they are very distressing. I am constantly having to run out into the garden." I said, "Why?" He said, "I am a horticulturist, and I am very fond of shrubs, and I see gipsies among them." I said, "I do not see any gipsies." He said, "Nothing will satisfy me that I do not see gipsies. When I open my eyes in the morning, I see gipsies using my tooth-brush. I bound out of bed, but no gipsies are there then." Of course, this was a very marked disorder, but nothing was done with regard to him until the following occurred: he went down to his office one Bank Holiday and got out a large quantity of very valuable securities from his safe, and he spent the morning looking through them. Then he had a visual hallucination. He thought he saw his son in the next office, and so he said, "Just put those things away," and went home. The son happening to go into the office later in the day, knowing his father had been there, found tens of thousands of pounds' worth of securities lying about loose in the office. That man, as a result of his hallucination, ran a great risk of losing valuable property. Therefore it was found absolutely necessary to put his property under the control of others.

And so, I think, it becomes evident that you may get any form of mental disorder associated with brain-decay, but the fact should not be forgotten that certain special symptoms are very characteristic. Perhaps the defect of memory is to be looked upon as the most characteristic of all. And a very useful subject of study would be to make a careful comparison between the defects of memory in cases of old age, as such, and in general paralysis. There is, of course, one common factor in all such cases as I have been considering, and that is that the mental condition depends very much on inheritance. In connection with this fact it may be observed that there are some individuals who seem as if wound up to go a certain time. Some young patients seem only capable of going like a twenty-four hour clock, and then have to be wound up again, if anything good has to be done at all. There are some who are wound up and go longer, but they always break down in the same way. There are females who break down on the nervous side, as life goes on; who lose control, as seen in mania and various hysterical conditions, leading to indecent exposures and improper relations. You know the proverb about the old man in marrying digging his own grave. He marries from the erotism of decay. It is not the marriage which kills him, for one has known cases of that kind in which a marriage between an old man and a young woman has never been consummated, and the old man has died within a short time of the wedding. All these things have certain roots in the neurotic heredity of the individual.

Finally, I ask you to remember that the most characteristic forms of mental disorder are (1) simple dementia, marked chiefly by loss of memory for recent events (for which patients little or nothing can be done). (2) There is loss of higher control, as evidenced in maniacal outbursts, and unfortunate cases of indecent exposure and the like. (3) Special attention is due also to characteristic cases of senile melancholia of the hypochondriacal type, which are frequently associated with ideas of obstruction.

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THE TREATMENT OF CHRONIC SUPPURATIVE OTITIS.

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It may still be said that the treatment of otorrhœa is often unsatisfactory. The frequency of mastoid operations is in itself an indication of past neglect or of failure to cure before implication of the bone. The serious results of this common trouble are not generally recognised. In every case there is some impairment of the general health; in many cases there is seriously-lowered vitality; and not a few end in sudden death. In some instances the bone becomes involved; pain may then be experienced of great intensity and persistence. These are facts; yet more or less wilful neglect is not uncommon, and patients are told they will grow out of it, or that it is dangerous to meddle with the ear.

In 1895 Mr. W. Macewen challenged the Otological Section of the British Medical Association with the failure of aurists to cure otorrhœa, and at the time little exception was taken to his statement. His advocacy of radical operations has found many supporters, but I noticed at the Otological Congress in 1899 that more than one member spoke of unsatisfactory results with the chisel. But however necessary such measures may be in the presence of extensive bone-disease, they are unnecessary and improper in the absence of deep implication of the bone. There is a simpler way of curing otorrhœa.

For failure in the past there are certain excuses. The account of the treatment of this important disease in most of the works on Otology was certainly inadequate, and the advice given has rather served to restrain medical men from working on sensible lines and from applying the principles of Listerism in the treatment of an infected and fairly tolerant cavity. "A little gentle syringing with warm water" was the advice given in one great work of surgery. On the other hand I have seen an article by an otologist denouncing the use of the syringe.

The intrinsic difficulties are sufficiently great, and these are increased when the meatus is obstructed and when there is only a small perforation in the membrana tympani. Even with a large perforation it is not always easy to disinfect a running ear, but much more may be effected by antiseptic irrigation properly carried out than some surgeons seem to think. What, however, if obstruction prevent efficient irrigation? The question of the advisability of removing the membrane with the malleus and perhaps incus, and of curetting the middle ear and attic, has been exercising my mind for some time. The advantages to be gained were obvious. What were the risks? The possibility of causing great pain seemed real enough, but experience has shown me how tolerant of interference these diseased ears usually become, especially after irrigation has been practised for some days. As for graver complications, I believed that a careful performance of this operation with subsequent irrigation would not prove unduly hazardous, and was a far safer proceeding than letting the disease run its course or than a so-called radical operation. For the past two years, I have performed this operation in suitable cases and am well pleased with the results. Combined with irrigation, "otectomy," if I may so term it, is of great assistance in cases where obstruction exists. The membrane should be cut out as completely as possible, especially in the case of children; with the membrane I nearly always remove the malleus and in some cases the incus. Granulations must be scraped out. I am strongly opposed to the opinion that granulations in these infected ears should not be interfered with. I have never known any trouble follow their removal, which certainly expedites the cure. Some authorities appear to regard them as a safeguard. I have nearly always found them diseased in these cases, and find a much surer safeguard in antiseptics. In cleansing the attic, I am sometimes assisted by a little instrument which has been made for me by Messrs. Wood of York. It consists of a flexible, slender steel rod ending in a roughened knob. This may be introduced into the attic through a silver tube shaped like a small Eustachian catheter. The curve of the catheter directs the end of the rod, which may be clothed with medicated wool, into the upper part of attic. The catheter itself is of use in directing the irrigating fluid into the accessory cavities. After the operation the ear is well irrigated with an antiseptic solution of silico-fluoride, or acetozone, which is less stimulating. Then after drying, I usually blow into the ear a little traumatol. This is a very light and finely-divided powder, and will penetrate further than any spray.

I have found the operation gives rise to very little disturbance in the majority of cases. I have not had any serious complication in any of my cases, if we except temporary facial paresis, which has twice occurred. There has been in most cases a remarkable absence of severe pain, especially in cases that had previously undergone a course of irrigation. On the other hand, I have had more than one case in which the operation gave great and immediate relief to pain.

With regard to the possibility of the operation affecting the hearing, about which patients are always anxious, it is of course impossible to give any assurance. I can only say that in no case have I noted any marked diminution in hearing, and that in some the hearing has certainly improved.

As regards general results, I have found the operation, as rendering possible efficient irrigation of the diseased cavities, of the greatest assistance. As I only operate in cases where there is a difficulty in irrigating, I have not a large number of notes, but the cases I have operated on furnish support to my belief that chronic suppurative otitis is generally curable by treatment through the meatus, and that such cases should not be allowed, through neglect, to go from bad to worse, nor subjected unnecessarily to the severe ordeal of a so-called radical operation.

CASES.

Case 1.—E. J., aged 33, has suffered from double otorrhœa for 28 years. She has attended a special hospital for some months without relief. When admitted to Coventry Hospital in December, 1902, both ears were full of a very offensive discharge. On December 13 I removed the greater part of both membranes with the mallei, and cleaned out the middle ear and attic. There was little pain complained of after operation, but some giddiness. The ears were irrigated frequently with solution of silico-fluoride, after which traumatol was blown in. The patient made a steady recovery. When last seen in May, the ears had been dry and sweet for some weeks, and presented a perfectly healthy appearance. This patient had been extremely deaf. There was some improvement in the hearing.

Case 2.—H. L., aged 20, has had a discharge from the right ear since infancy. There has been much pain of late. He had been under treatment elsewhere, but found that "syringing was not the least good." On November 1 I removed the greater part of the membrane and cleaned out the cavities. I also scraped a carious cavity in the bone. Irrigation was carried out twice daily with silico-fluoride. During a rather long after-treatment, use was made of formalin, protargol and eucalyptus. Irrigation was materially assisted by my little catheter. The patient could only attend twice a week, but carried out irrigation at home. The operation was well borne and relieved the pain at once; no subsequent return of pain was complained of. The discharge gradually lessened, and when the patient was last seen in August, the condition of the ear remained satisfactory.

Case 3.—A. R., aged 33, had been for many years under occasional treatment by specialists without relief. A course of salufer irrigation had given great relief, but a focus of inflammation remained round the malleus, until last year I removed the bone with the remains of membrane. There was no pain or disturbance, and the ear was healed in three weeks. She has been quite well since.

Case 4.—L. D., aged 30, was sent to me by Dr. Milner Moore, complaining of an occasional discharge from the left ear, which had lasted for several years. Pain began to be noticed two years ago; this was referred chiefly to the front and upper part of the ear, but there was tenderness over the mastoid. This lady was in a feeble state of health. As irrigation failed to cure after some months trial, I removed the malleus and remains of the membrane last March. Rough carious bone was felt in the middle ear and attic, part of the outer wall of which I removed. The operation gave rise to no special disturbance, but the condition was not materially improved. Pain in front of the ear is still complained of, and there is occasionally a rather copious discharge, which is not now offensive. The mastoid tenderness has quite disappeared. The middle ear looks healthy. I am inclined to think the discharge comes through a fissure in the wall of the middle ear.

Case 5.—A. L., aged 29, had suffered from double otorrhœa for 22 years, and had attended a special hospital for nine months, when three operations of some kind were performed; she was also an in-patient at another hospital for four months on account of her ear. She is a very feeble person, deaf, with both ears very offensive. On April 26 I removed some granulations from each ear, but did nothing further, at the request of the anæsthetist. On May 9th I removed the malleus and part of membrane from the left ear. No pain was complained of after the operations. On May 22 great improvement was noted. Irrigation was ordered to be continued, and a two-per-cent. solution of protargol to be poured into the left ear each day. On June 5 both ears were dry and glistening. She could hear a watch one foot away with the right ear, four inches off with the left; conversation easily. She has remained well, her general health being much improved.

Case 6.—Mrs. B., aged 28, was sent to me by Dr. Leslie Callaghan. She had had discharge from the right ear as a child and occasionally since. There had been much pain in the ear of late, and swelling below the ear in the mornings. A small perforation was visible in a swollen, unhealthy-looking membrane. On June 11 I removed the membrane. No pain was complained of after the operation. The ear settled down quickly, and on June 30 was dry and sweet. There has been no return of trouble. Mrs. B. says her hearing is much better.

Case 7.—E. R., aged 20, has had a discharge from each ear for about six months and is very deaf. On May 3 I removed a large polypus from the left ear and did an otectomy on the right ear, removing the membrane with the malleus and incus. The operation caused hardly any pain. Irrigation was ordered three times a day with a weak salufer solution. On May 22 it was noted that hearing had improved considerably; she could now hear a watch a foot off on each side. The discharge is now lessening and is not offensive. On June 5 both ears were dry and sweet, and have remained so.

Case 8.—Miss S., aged 33, had had a discharge from the right ear since infancy, with occasional pain. The ear was very offensive and revealed a minute perforation. On June 30 I removed the membrane and malleus. Very little pain occurred after the operation, but there was severe sickness after the chloroform. Rapid progress was made after the first day or so. The ear was dry and sweet before the end of July. Seen in September, the ear was perfectly healthy. Miss S. says the hearing of the affected ear has improved.

Case 9.—Miss C., aged 25, was sent to me by Dr. Banks Price. She had had bad earache in childhood with many "gatherings" in the left ear, and was very deaf on that side. She was sent to me on account of deafness and severe pain. The membrane was unhealthy-looking with a small perforation high up behind the malleus. I removed the membrane on June 25. Severe pain followed the operation, but was subdued by leeches and opium. The pain came on again on several occasions after irrigation. I had to give up salufer and substitute acetozone, which was better borne. In other respects the ear progressed favourably, and all pain and discharge had ceased by the end of July. The ear remains healthy. Miss C. told me recently that the hearing was much better.

Case 10.—L. P., aged 27, was admitted on May 1 to the Coventry Hospital for offensive discharge from both ears, of long duration. The left ear was somewhat swollen, and there was tenderness over the mastoid. Two leeches were applied and irrigation commenced. On May 22 it was noted that the right ear was much better. On May 24 I removed the remains of the left membrane. The posterior wall of the bony meatus was carious and quite soft in one place. After the operation a good deal of pain was experienced. A paralysis of the left facial nerve, which had been slight previous to the operation, was much more marked. The ears, however, gradually became dry and sweet, and their condition is still satisfactory (October 9). Under galvanism the condition of the facial nerve rapidly improved, and it has now quite recovered. Hearing of the left ear is good.

Case 11.—H. B., aged 15, has had a discharge from the right ear for four years, and had been under treatment at a special hospital without relief. He was a very anæmic boy with weak pulse and some gastric catarrh. A copious discharge, of evil odour, escaped through a small perforation. On

November 16 I removed the membrane with the malleus. This case got well rapidly; there was no discomfort after the operation. The ear became sweet in a very few days, and all discharge had ceased at the end of three weeks. As the boy was kept in hospital on account of his general condition till January 12, one was able to keep the ear under observation. There has been no relapse.

As space is limited, I will only mention two cases of children operated on. Otorrhœa in young children has special features about which much might be said, but I only wish to refer here to two peculiarities—(1) the tendency to a persistence of discharge after the ear has become sweet, and a consequent danger of reïnfection, and (2) the rapid repair of the membrane frequently seen after partial removal. I have found that children tolerate interference well; in their case removal of the membrane should be as complete as possible, if one wishes to make a permanent opening. Remains of the membrane after use of the knife should be removed by scissors or punch.

Case 12.—R. D., aged 5, was a delicate child suffering from otorrhœa. Last year I removed the greater part of membrane without occasioning any pain or disturbance. The ear became sweet after a course of irrigation, and the discharge lessened. The house-surgeon having reported that there had been no discharge for a fortnight, the child was sent home. On later examination, however, I found that there was still discharge, but that the membrane had almost completely closed up. Irrigation was again started; but as no real improvement followed, I have this month again removed the membrane as completely as possible. The child was quite unaffected by the operation. He is still under treatment.

Case 13.—P. C., aged 7, has had left otorrhœa for twelve months. An offensive discharge escapes though a small perforation. On July 24 I removed a large portion of the membrane. The child made no complaint of pain, and the ear quickly got well; it was quite dry and sweet when she left in August. I saw her on September 18, and found that the membrane had quite healed. Hearing good on the affected side.

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CLINICAL EXAMINATION OF THE GASTRIC CONTENTS.

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It is proposed to restrict the present paper to the question of the chemical examination of the contents of the stomach. It is not only in cases of carcinoma or other severe organic lesion of the organ that investigations of its contents are of value; in those widespread lesser ailments which are vaguely grouped together as Dyspepsia, the condition of the gastric contents forms an even more important subject of study. It is to be regretted that such examinations are so seldom carried out. The difficulty of deciding when to prescribe alkalies, and when acids, might be lessened if the nature of the gastric juice were first investigated in each individual patient. In many cases a normal juice would be found; in these neither alkalies nor acids might be needed, and treatment would be directed to cleansing or replacing the teeth, modifying the diet and the patient's general mode of life, stimulating the muscular wall of the stomach, or regulating the action of the bowels. In others an excess of free hydrochloric acid will be found, and treatment by alkalies and perhaps an increased proteïd diet indicated. In some of this class the acid would be in excess as early as half an hour after food, in others not until an hour or more later, and the application of a few easy tests would decide the best time for administering the alkali. In other people there would be deficiency of hydrochloric acid, or the gastric juice might even be quite alkaline, so that mineral acid must be artificially supplied, to some in small, to others in larger doses. The stomach might be found to contain food long after it should have emptied itself; drugs such as nux vomica would then be indicated, in order to stimulate the non-striped muscle of its walls. The pepsin of the gastric juice is less often defective than is the amount of acid; but occasionally it is found wanting, and then its place may be supplied by artificial pepsin, or the need for it diminished by restricting the proteïd portion of the diet. Without examination of the secretion of the stomach, to determine first whether it be healthy or not; and secondly, if found unhealthy, to determine in what respect the departure from health has taken place; the treatment of dyspepsia must surely be haphazard and uncertain.

METHODS OF OBTAINING AND TESTING GASTRIC CONTENTS.

There are two main difficulties in carrying out such examination. The first—the less important of the two—is that the medical man has not the time for it, or to be more accurate, it is not so much that he has not the time, as that he has not the reägents and the apparatus ready. Were these to hand, the chief points could well be tested for in less than a quarter of an hour. The essential apparatus is a spirit-lamp, a metal dish for boiling water in, a few small evaporating basins, a funnel, some fine muslin, test-tubes, and litmus-paper. The essential test-solutions are Congo red, Günsberg's reägent, and Uffelmann's reägent; these and one or two alternative tests are described below.

The second difficulty is that gastric contents are not readily obtainable in private practice. This is the more serious obstacle. A patient suffering from a dilated stomach or from gastric carcinoma vomits spontaneously, with the result that the vomit is usually tested in such cases. A patient who has dyspepsia may never vomit, so that artificial means must be employed to provide the fluid to be examined. Unless the patient suffered very greatly, the proposal to use the uncomfortable stomach-tube would often be refused. Not many persons, on the other hand, who had endured the discomforts of dyspepsia for any length of time, would object to an emetic, if they understood the object with which it was given. Thirty grains of zinc sulphate may be administered for the purpose, and the stomach-contents easily obtained.

Provided it be known exactly what food has been taken last, there is no real necessity to give a "test" meal. Some foods take longer than others to be digested, and therefore, according to the character of the meal, so will the time vary for the gastric juice to reach its point of greatest activity. After proteïd food a longer time elapses before free hydrochloric acid can be detected than after carbohydrate or fat, because the acid first secreted becomes loosely attached to proteïd molecules as "combined" hydrochloric acid, and it is only after the proteïd has been satisfied in this way that a surplus can be detected as "free" acid. The average time required for this process is three-quarters of an hour, and in the meanwhile the starch-digesting ferment of the saliva continues its activity in the stomach. When gastric digestion has reached its height, there should be 0·2 per cent. of free hydrochloric acid. This point is attained earlier when carbohydrates alone have been eaten, later when proteïds have been taken too; so that it is usually convenient to give a "test" meal of known composition, and to administer the emetic at a definite interval afterwards. The following are examples of test-meals, and the times given for recovering them are those at which the gastric juice should have the full characters of health :—

Ewald's breakfast. 8 o'clock :—

White bread - -	70 grammes.
Weak tea - - -	300 cc.

Empty the stomach in 1 hour.

Klemperer's breakfast. 8 o'clock :—

White bread - -	70 grammes.
Milk - - -	500 cc.

Empty the stomach in 2 hours.

Germain See's lunch. 11 o'clock :—

White bread - -	150 grammes.
Minced meat	80 grammes.
Cold water - -	300 cc.

Empty the stomach in 2 hours.

Riegel's dinner. 2 o'clock :—

Soup - - -	100 cc.
Beef-steak - -	60 grammes.
White bread - -	50 grammes.

Empty the stomach in 5 hours.

In practice Ewald's breakfast is the most serviceable; and there is no objection to toasting the bread, or to putting a little butter on it.

EXAMINATION FOR HYDROCHLORIC ACID.

Having obtained the vomit, the chief point to examine is, as a rule, its acidity. First strain out the particles of undigested food by throwing the whole on to a piece of muslin supported in an ordinary funnel. It is not advisable to use filter-papers for this purpose, because there are at least four factors which go to make up the total acidity, namely :—

1. Acid salts, chiefly acid sodium phosphate ;
2. "Combined" hydrochloric acid ;
3. Organic acids, chiefly lactic, with traces of acetic and butyric ;
4. "Free" hydrochloric acid ;

and it is found that filter-papers allow some of these to pass through more rapidly than others, with the result that the filtrate has a different acidity to that of the original vomit.

Next test with neutral litmus-paper ; reddening of the latter will indicate acidity, but will not show to which of the above four factors this acidity is due. Acid salts or "combined" hydrochloric acid redden litmus-paper as readily as do organic or "free" mineral acids. If, however, the test-paper be not reddened, then a pathological condition is at once found, namely, the existence of a neutral or alkaline medium, in which pepsin is unable to digest proteïd.

If the litmus-test be positive, next test with Congo red. The solution is made up as follows :—

Congo red - - - 0·1 gramme,
Water - - - - 100 c.c.,

and it is convenient to make test-papers by dipping filter-paper into the solution, allowing it to dry, cutting it into strips, and storing these ready for use. Pour a drop of the strained vomit on to one of the red strips, hold it up to the light, and observe if any change of colour has occurred. Acid salts and "combined" hydrochloric acid will produce no change ; weak solutions of lactic acid will darken the Congo red to a deep brown ; strong lactic acid, or dilute free hydrochloric acid, will turn it a deep blue.

Should the Congo-red test indicate the presence of free acid, one or other of the following tests for free hydrochloric acid may be employed :—

Günsberg's Test.—The reägent is made up as follows :—

Phloroglucin	2 grammes.
Vanillin	1 gramme.
Rectified spirit	100 c.c.

The bottle in which it is kept should be well stoppered, and is best stored away from bright daylight. The solution keeps good for many weeks, but slowly loses its delicacy as a test, so that fresh quantities must be made from time to time. One of the best ways of applying the test is as follows :—Boil some water in a shallow pan, to serve as a water-bath ; float a clean dry porcelain evaporating-dish upon the water, and into the dish put *three drops* of Günsberg's reägent and *three drops* of the strained gastric contents ; as the mixture slowly dries it assumes a beautiful rose-red colour, first around the edges and finally all over the dried residue, if free hydrochloric acid be present. Further heating causes this rose-red colour to disappear again, but if the dish be taken off the water-bath and cooled, the colour will remain for several hours. There are three chief sources of error to avoid in performing the test, and unless attention be paid to them free hydrochloric acid may be missed. The first is overheating ; to avoid this the water-bath is used ; application of a flame directly to the evaporating dish often causes the contents to char without giving the pink colour. The second is using too large a bulk either of the reägent or of the strained vomit ; three drops of each are plenty. The third is using unstrained vomit ; the presence of solid particles of food will often vitiate the test, apparently because the heated hydrochloric acid is used up in acting upon this food rather than upon the phloroglucin and vanillin.

The test properly applied is very easy ; it takes but a few minutes, and is very delicate. A similar rose-red colour is given with any free inorganic acid, such as nitric acid or sulphuric ; it is also given with *strong* solutions of organic acids, such as acetic or lactic. Unless the patient have just swallowed a quantity of vinegar, for example, there is never sufficient organic acid in a vomit to give the test ; nor is any other mineral acid than hydrochloric likely to be present ; the rose-red colour is not given by "combined" hydrochloric acid ; therefore Günsberg's is one of the best tests for the free acid. Moreover, the depth of the rose-red colour is greater the more free hydrochloric acid there is present, so that, if exactly three drops both of strained vomit and of the test solution be always used, the resulting colour affords an approximate idea as to whether the amount of free acid is deficient, normal, or excessive.

The following are alternative tests, which may be used either in addition to, or in place of, Günsberg's:—

Boas' Test.—The reägent is made up as follows:—

Resorcin	5 grammes.
White sugar	3 grammes.
Rectified spirit	100 c.c.

It is more stable than Günsberg's solution, and is also cheaper. The test is performed in exactly the same way as Günsberg's, using an evaporating-basin on a water-bath, three drops of reägent, three drops of strained vomit, and watching for a rose-red colour in the residue as it dries. The disadvantage of the test is that the red colour appears more slowly. Often the test seems to be giving a negative result, when warming the dried residue for another ten minutes will bring out the characteristic rose-red. To a busy man this is a very real objection. In other respects the test is very similar to Günsberg's.

The Tropæolin test.—Tne reägent is made up as follows:—

Methyl orange	0·2 gramme.
Rectified spirit	25 c.c.
Distilled water to	100 c.c.

The solution keeps good indefinitely, even in broad daylight. To perform the test, place one drop of the solution upon a white porcelain slab, spread it out in a thin layer over an area about equal to that of a penny, and dry it at 40° C. If there be a Bünsen flame available, this may be done by placing the slab upon the bench close to the foot of the burner; if a spirit-flame be used, the slab may be directly warmed until it feels to be about body-temperature. When the tropæolin has dried, put a single drop of the strained vomit upon the centre of the yellow stain; if free hydrochloric acid be present, a beautiful violet colour appears, and becomes intensified as the fluid dries again at body-temperature. It is a very easy and pretty test; but if a considerable quantity of lactic acid be present, it will give the colour too. Seldom, however, is sufficient lactic acid present in the gastric contents to give the reäction, so that the test is usually held to be good proof of the presence of free hydrochloric acid.

The Methyl-Violet Test.—The solution is made up as follows:—

Methyl violet - - -	0·1	gramme.
Distilled water - - -	100	c.c.

The reägent keeps well. It is an easy test, and is applied thus: One drop of the methyl violet is placed upon a white porcelain surface, and close to it one drop of the strained vomit. The two drops are made to coälesce, and the colour watched at the point where they mix. A change from violet to greenish-blue occurs if free hydrochloric acid be present, but there are some conditions in which sufficient lactic acid may be present to produce a similar change.

TEST FOR LACTIC ACID.

The test employed for detecting lactic acid is known as *Uffelmann's*. The solution required is best made fresh each time by adding a few drops of liquor ferri perchloridi to half a test-tubeful of carbolic acid (1 in 20). A purple-blue colour results. On adding the strained vomit to an inch of this in a test-tube the blue colour disappears, and if lactic acid be present, the liquid becomes canary-yellow. If free hydrochloric, but no lactic acid be present, the blue colour is discharged entirely, the resulting liquid being colourless; but not infrequently a doubt as to the colour arises, because a precipitate of phosphates comes down. It is the *solution* which must be canary-yellow to indicate presence of lactic acid; therefore in all cases the mixture of Uffelmann's reägent and of strained vomit must be filtered, and the colour of the *filtrate* observed. The importance of filtering will be at once seen if, to a little Uffelmann's reägent in a test-tube, some ammonia-solution be added; a bright-yellow colour replaces the blue, but, on filtering, the filtrate is colourless; all the yellow colour in this case is in a very fine flocculent precipitate. It must also be remembered that there is a certain yellowness about the strained vomit itself, so that the filtrate which results from Uffelmann's test must be yellower than the original stomach-contents before lactic acid can be said to be present.

It may be well here to summarise shortly the steps to be employed in testing the acidity of gastric juice:—

(*a*) First strain the vomit through fine muslin, and not through filter-paper.

(*b*) Test with neutral litmus-paper; a red reäction indicates acidity due to one or more of the following: (1) acid sodium phosphate; (2) combined hydrochloric acid; (3) lactic acid; (4) free hydrochloric acid.

(*c*) Test with Congo red; a blue reäction indicates lactic acid or free hydrochloric acid; a brown reäction is indeterminate; neither acid sodium phosphate nor combined hydrochloric acid alter the red colour at all.

(*d*) Test with Günsberg's reägent; a rose-red colour indicates free hydrochloric acid; this is the most satisfactory test for the purpose, though Boas' test, the tropæolin test, or the methyl-violet test, may be employed as alternatives.

(*e*) Test with Uffelmann's reägent; a light-yellow colour of the filtrate indicates lactic acid.

In regard to the interpretation of the results found, stress has already been laid upon the importance of knowing how long previously the last food was taken, and what proportion of proteïd it contained. Absence of free hydrochloric acid for three-quarters of an hour after food is physiological. If a test-meal has been given, and recovered in the prescribed time, Günsberg's test should readily give a fine rose-red reäction. If this be not so, either an attempt should be made to supply free hydrochloric acid by the mouth; or the proteïd portion of the diet should be very finely divided before it is swallowed. The stomach is loth to allow large solid particles to pass through the pylorus, and yet it cannot, in the absence of free hydrochloric acid, disintegrate pieces of solid meat. If, on the other hand, Günsberg's test show that free hydrochloric acid appears too early—that is to say, within three-quarters of an hour after a meal—the action of the starch-dissolving ferment of swallowed saliva is checked too soon, and the carbohydrates, which the gastric juice cannot digest, remain as offending solid lumps. Attempts should be made to prevent the appearance of free hydrochloric acid quite so soon by giving sodium carbonate, or other alkali, at a period of 10, 15, 20, 25, 30 or 35 minutes after food, according to the time at which the free acid is first present. The same end may be to some extent attained by giving more proteïd at meal-times; such additional proteïd must first be satisfied before any surplus hydrochloric acid will be "free." There are two factors which limit the latter form of treatment; the first is, that proteïd food more than any other stimulates the stomach to increase the secretion of its juice; and the second is that the kidneys may suffer if too much work be thrown upon them in the elimination of nitrogenous waste products.

Free hydrochloric acid may not appear unduly soon, and yet it may continue to be secreted to an abnormal extent. Instead of reaching 0·2 per cent. and not increasing further, it may rise to as much as 0·3 per cent. or 0·4 per cent., in cases of hyperacidity and gastric ulcer. Perhaps the hyperacidity is one factor in the production of gastric ulcer; the frequent association of the two conditions is suggestive. Even if this be not so, it is desirable to check such hyperacidity if possible, and only by examination of the gastric contents can this be done methodically. The depth of the rose-red colour given by Günsberg's test affords a means of judging when free hydrochloric acid is present in excess. This will not be immediately after a meal; and the time when hyperacidity begins will vary in different people. In some an alkali will be indicated one hour after meals, in others an hour and a half after, in others two hours, and so on, examination of the gastric contents affording the ideal criterion in each individual patient.

DIAGNOSTIC VALUE OF THE EXAMINATION.

In the present paper examination for free acid is urged chiefly from the point of view of treatment. It is also said to have a diagnostic value; in cases, for example, of gastric carcinoma much stress is often laid upon the absence of free hydrochloric acid. The writer is more than doubtful of the value of this as a diagnostic test. In the first place the site of a stomach-cancer does not usually coïncide with that of the oxyntic cells; the latter are in the cardiac portion of the stomach, the former usually at the pylorus. In the second there are a very large number of conditions in which, time and again, absence of free hydrochloric acid from the stomach-contents has been demonstrated. In many febrile conditions this may be so; for instance, in patients suffering from enteric fever or from influenza. In chronic congestion of the stomach it is sometimes so (for example, in patients suffering from failing heart); sometimes in Bright's disease, again, and in many cachectic conditions. Indeed, in those cases of gastric carcinoma in which free hydrochloric acid has been absent, it seems not improbable that the cancer may have caused cachexia, and that the cachexia rather than the locality of the growth has been the immediate cause of the deficiency in acid. Free hydrochloric acid has been present in more than one case of gastric carcinoma, and many a sick patient who has no cancer of the stomach is found to have no free hydrochloric acid in the vomit. With ill-health all the tissues and secretions of the body suffer, and amongst them the gastric juice. Bearing in mind that gastric ulcer is often associated with hyperacidity, it may be allowed, perhaps, that when the diagnosis lies solely between that of gastric ulcer and that of gastric growth, great deficiency or absence of free hydrochloric acid is in favour of the latter, whilst hyperacidity is in favour of the former; even in such cases, however, the diagnostic value of the test is much restricted, for in many the free acid is neither absent nor in excess.

The following are the results obtained on testing a large number of consecutive vomits in the wards, taken indiscriminately. The chief lesson to be learned from them is that, when a patient is really ill, the gastric secretion is unfit for heavy work, and the diet must be of the simplest. This is not likely to be forgotten when the patient has a fever, but in cases such as heart-disease with signs of failure it is more likely to be overlooked. An overloaded stomach is not infrequently the direct cause of death in such cases.

RESULTS FOUND ON TESTING THE VOMIT OF 38 PATIENTS SUFFERING FROM VARIOUS DISEASES.

In all cases vomiting was more than three-quarters of an hour after food; and the diet was either milk, or bread-and-butter and tea.

Affections of the Stomach.

No. of Case.	Diagnosis.	To Litmus.	To Congo Red.	To Uffelmann's Test.	To Günsberg's Test.	Lactic Acid.	Free Hydrochloric Acid.
1	Fibrous stricture of pylorus and dilated stomach.	Alkaline.	No.	No.	No.	No.	No.
2	Simple stricture of pylorus and dilated stomach.	Very acid.	Blue.	Canary-yellow.	No.	Excess.	No.
3	Old cicatrised ulcer; dilated stomach.	Acid.	Blue.	Pale-yellow.	Rose-red.	Present.	Present.
4	Simple dyspepsia -	Acid.	No.	No.	No.	No.	No.
5	Carcinoma of stomach	Acid.	Deep-brown.	Canary-yellow.	No.	Excess.	No.
6	Carcinoma of stomach	Very acid.	Blue.	No.	Rose-red.	No.	Present.
7	Carcinoma of stomach	Acid.	Deep-brown.	Canary-yellow.	No.	Excess.	No.
8	Carcinoma of stomach	Acid.	Blue.	No.	Rose-red.	No.	Present.

Febrile Affections.

9	Enteric fever, relapse -	Acid.	Blue.	No.	Rose-red.	No.	Present.
10	Enteric fever - -	Neutral.	No.	No.	No.	No.	No.
11	Enteric fever - -	Acid.	No.	No.	No.	No.	No.
12	Influenza - -	Acid.	No.	No.	No.	No.	No.

Pulmonary Affections.

13	Fibroid lung and bronchiectasis.	Acid.	Deep-brown.	Pale-yellow.	No.	Present.	No.
„	Do., another day	Strongly Acid.	Blue.	No.	Rose-red.	No.	Present.
14	Phthisis - - -	Acid.	Brown.	Pale-yellow.	No.	Present.	No.
15	Phthisis - -	Acid.	No.	No.	No.	No.	No.
16	Bronchiectasis -	Acid.	Brown.	Canary-yellow.	No.	Excess.	No.
17	Phthisis; Spinal caries; lardaceous disease.	Neutral.	No.	No.	No.	No.	No.
18	Phthisis; lardaceous disease.	Neutral.	No.	No.	No.	No.	No.

Nervous Affections.

19	Myoclonus lumbalis et cervicalis.	Very acid.	Blue.	No.	Rose-red.	No.	Present.
20	Cerebral tumour, on several occasions.	Acid.	No.	No.	No.	No.	No.
„	Do., on one occasion	Alkaline.	No.	No.	No.	No.	No.
„	Do., on one occasion	Very acid.	Brown.	Canary-yellow.	No.	Present.	No.
„	Do., on one occasion	Very acid.	Blue.	Canary-yellow.	Rose-red.	Present.	Present

Heart-Affections.

No. of Case.	Diagnosis.	To Litmus.	To Congo Red.	To Uffelmann's Test.	To Günsberg's Test.	Lactic Acid.	Free Hydrochloric Acid.
21	Aortic and mitral, with failure on seven different occasions.	Acid.	Brown.	Canary-yellow.	No.	Present.	No.
„	Do., on one occasion	Acid.	Blue.	Canary-yellow.	Light rose-red.	Present.	Present.
22	Heart failure - -	Strongly acid.	Brown, slight.	No.	No.	No.	No.
23	Aortic and mitral, almost compensated.	Strongly acid.	Brown.	Canary-yellow.	No.	Present.	No.
24	Valvular disease and failure.	Alkaline.	No.	No.	No.	No.	No.

Various other Affections.

25	Exophthalmic goitre -	Acid.	No.	No.	No.	No.	No.
26	Exophthalmic goitre -	Strongly acid.	Blue.	Canary-yellow.	Rose-red.	Present.	Present.
27	Perihepatitis and ascites.	Acid.	No.	No.	No.	No.	No.
28	Cirrhosis of liver, ascites, cholæmia.	Strongly acid.	Blue.	No.	Rose-red.	No.	Present.
29	Cirrhosis of liver -	Acid.	Blue.	No.	Rose-red.	No.	Present.
30	Acute tubal nephritis -	Just acid.	No.	No.	No.	No.	No.
31	Diabetes mellitus -	Acid.	Brown.	Light-yellow.	No.	Present.	No.
32	Tubercular peritonitis	Alkaline.	No.	No.	No.	No.	No.
33	Appendicitis - -	Alkaline.	No.	No.	No.	No.	No.
34	Appendicitis -	Acid.	No.	No.	No.	No.	No.
35	Carcinoma of cervix uteri and double pyonephrosis.	Very acid.	Blue.	Canary-yellow.	Rose-red.	Present.	Present.
36	Carcinoma of colon -	Acid.	No.	No.	No.	No.	No.

In regard to the lactic acid, this is a normal constituent of the gastric contents in the earlier stages of digestion. During the first three-quarters of an hour, before any free hydrochloric acid is present, the lactic acid increases rapidly in amount, particularly when the diet contains milk. When free hydrochloric acid appears, the formation of lactic acid diminishes and soon ceases ; for a time both acids are present together ; then the lactic acid disappears, so that at the height of digestion, with 0·2 per cent. of free hydrochloric acid, lactic acid can no longer be detected. It is therefore difficult, in most cases, to draw any conclusions from the lactic-acid test, unless there be something very much the matter. Absence of free hydrochloric acid may allow much acid fermentation in the stomach, with production of an obvious excess of lactic acid. Acid dyspepsia is often of this nature. It may occur in any diseases in which free hydrochloric acid is absent, and amongst these in gastric carcinoma. It is not distinctive of any particular affection, but indicates the necessity of being careful with the diet, of assisting the pepsin by giving dilute mineral acids by the mouth, and perhaps of inhibiting the action of microörganisms by such drugs as glycerine of carbolic acid or sulphocarbolate of sodium.

QUANTITATIVE ESTIMATION OF ACIDITY.

In some cases it is important not only to test, but also to estimate, the acidity. As has been mentioned, the depth of rose-red colour with Günsberg's test affords a rough idea of the amount of free hydrochloric acid present; but in difficult cases of acid dyspepsia, or of gastric ulcer with hyperacidity, greater accuracy is needed. It is easy to estimate the total acidity, but less so the acidity due to free hydrochloric acid. In either case a decinormal soda-solution is required, and this is made by dissolving 4 grammes of fused caustic soda in distilled water up to 1,000 c.c.

To estimate the total acidity, 100 c.c. of the strained vomit may be measured into a beaker; 10 drops of phenol-phthaleïn solution added; and the beaker placed upon a white slab beneath a burette filled with decinormal soda-solution. The soda is added, a few drops at a time, until the yellow colour of the fluid in the beaker just changes to a definite light-pink. The volume of standard soda-solution required to do this contains the amount of caustic soda needed to neutralise all the acid salts, combined hydrochloric acid, lactic acid, and free hydrochloric acid in the 100 c.c. of vomit. The "acidity" of the latter may be expressed in terms of caustic soda, or a simple calculation will give it in terms of hydrochloric acid. It is best expressed in terms of caustic soda, because the total acidity is far from being all due to hydrochloric acid.

To estimate the proportion of the acidity that is due to free hydrochloric acid is not so simple. There are several methods for doing it accurately, but most are too complicated for clinical use. The following gives very approximate results, and is simple, though it takes perhaps half an hour to carry out. Float six clean evaporating-basins upon the water-bath, and into each put two drops of Günsberg's reägent. Number them 1, 2, 3, 4, 5, and 6. Measure 100 c.c. of strained vomit into a beaker, and add 5 c.c. of decinormal soda-solution from the burette. Stir well, and transfer two drops of the contents of the beaker to the evaporating-basin marked 1. Now add another 5 c.c. of decinormal soda-solution to the vomit in the beaker, stir well, and transfer two drops of the contents of the beaker to the evaporating-basin marked 2. Add another 5 c.c. of the soda to the beaker, stir, and transfer two drops to the basin marked 3; and so on. That is to say, after each addition of 5 c.c. of decinormal soda-solution to the 100 c.c. of vomit, do a Günsberg's test. Suppose evaporating-basins No. 1 and No. 2 give a good rose-red colour, whilst No. 3 gives a paler tint, No. 4 a very pale, and No. 5 none at all, it follows that 20 c.c. of decinormal soda-solution are just too little to neutralise all the free hydrochloric acid in the 100 c.c. of strained vomit, whilst 25 c.c. are just too much. Now repeat the process again; take four clean evaporating-basins to each of which two drops of Günsberg's reägent have been added; number them 1, 2, 3, and 4; float them on the water-bath; measure a fresh 100 c.c. of strained vomit into a beaker, add 21 c.c. of decinormal soda-solution, and put two drops of the beaker-contents into basin No. 1. Add another 1 c.c. of soda, and put two drops of the beaker-contents into basin No. 2; similarly with basins No. 3 and No. 4, after each addition of 1 c.c. of soda to the 100 c.c. of vomit. Suppose basin No. 1 give a faint pink colour, No. 2 a very faint tinge, and No. 3 none at all, it follows that 22 c.c. of decinormal soda-solution are just insufficient to neutralise all the free hydrochloric acid in 100 c.c. of strained vomit, whilst 23 c.c. are just too much; the mean between the two is the measure, in terms of caustic soda, of the acidity due to free hydrochloric acid.

If the total acidity be known, and also that due to free hydrochloric acid, the difference gives that due to acid sodium phosphate, to lactic acid, and to combined hydrochloric acid, together. There is no simple clinical method for estimating these separately.

ESTIMATION OF PEPSIN.

It is sometimes important to test the activity of the peptic ferment, though this is less often defective than is the hydrochloric acid. For this purpose discs of hard-boiled white of egg may be prepared. Boil an egg for twenty minutes, allow it to get cold, and then peel the white from the yolk, cut the former into strips, and punch out discs with a cork-borer. These discs may be kept in glycerine ready for use. To apply the test, take four test-tubes, number them 1, 2, 3, and 4, put 5 c.c. of strained vomit and 1 disc of egg white into each, and stand them in a water-bath heated to body-temperature. Add nothing further to No. 1. To No. 2 add 2 grains of dried pepsin. To No. 3 add 2 drops of dilute hydrochloric acid (B.P.). To No. 4 add 2 drops of dilute hydrochloric acid and 2 grains of pepsin. Leave them for an hour, and then observe which of the tubes show solution of the discs. If No. 1 does so, the gastric juice contains active pepsin, and is sufficiently acid to allow of its digesting proteïd. If No. 1 does not, but No. 2 does, the gastric juice is suitable for peptic activity but is lacking in pepsin. If No. 1 and No. 2 do not, but No. 3 does, then the gastric juice contains pepsin, but too little hydrochloric acid to allow it to be active. If No. 1, No. 2, and No. 3 do not, whilst No. 4 does, then the gastric juice is deficient both in hydrochloric acid and in pepsin. Should pepsin be found wanting, its place may be artificially supplied by prescribing one of the preparations of this ferment.

EXAMINATION FOR RENNIN.

In the gastric juice, particularly of children, another ferment, rennin, should be present. In its absence cow's milk is ill-digested, forming dense curdled masses in the stomach. It may readily be tested for as follows :—Neutralise 10 c.c. of strained vomit by adding two drops of phenol-phthalein as an indicator, and dilute caustic soda until a faint-pink colour appears ; add 10 c.c. of fresh cow's milk, previously boiled and allowed to cool nearly to body-temperature. If rennin be present, the caseïnogen of the milk will presently be precipitated in fine flakes. Should rennin be absent from the gastric contents, it will be necessary either to add barley or lime-water to the milk to ensure a finer precipitate of caseïn in the stomach; or to peptonise the milk before giving it; or to give rennet in the form of curds and whey; or even in some cases to replace the milk for the time being by some other food, such as albumen-water.

Estimations of acidity are rarely necessary in private practice; testing the acidity and investigating the ferments are far more valuable. It is a pity that the tests are so seldom applied. To examine a vomit for the nature of its acidity occupies but little longer time than does the proper testing of a specimen of urine. It cannot but be that the treatment of dyspepsia would be more successful were an emetic administered to the patient after the object in view had been explained, and were the prescription based upon the indications given by the clinical examination of the gastric contents.

THE QUESTION OF OPERATION IN CANCER OF THE STOMACH.

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So far as the treatment of malignant disease in general is concerned, there can be little doubt that the surgeon has ample reason for congratulating himself upon the achievements of recent years. The immediate risks of operation have been reduced to a mere fraction of what they formerly were, notwithstanding the fact that a more extensive procedure is always adopted; the healing of wounds is as speedy and as satisfactory as is possible; and the number of patients who are cured of their disease is a steadily-increasing one. The most exemplary instance of the change wrought in the treatment of malignant disease during the last 10 or 20 years is found in the case of the breast. The results of Halsted, Cheyne, and other surgeons would hardly have been deemed possible 20 years ago. But in striking and lamentable contrast with this general improvement we have to admit the entire, or almost entire, lack of any reasonable progress in the surgery of malignant disease of the stomach. Admitting to the full the difficulties that lie in the way of an earlier recognition of the disease, it must nevertheless be avowed that our results are not as satisfactory as they ought to be.

In discussing the causes of this, and the possibility of removing the reproach, the point to be first considered is the question of early diagnosis. Every surgeon knows by bitter and not infrequent personal experience that it is no uncommon event for him to be asked to see, with a view to operation, a patient with advanced malignant disease of the stomach, a patient whose abdomen contains a tumour readily palpable, or obvious even on inspection. In many cases surgery can offer no prospect to these patients; they can but drift, hopeless and helpless, to an inevitable end.

Malignant disease of the stomach begins in the great majority of cases in patients over 45 years of age, and its onset is not infrequently almost abrupt. A patient who has previously enjoyed good health begins unaccountably to suffer from heaviness, fulness, or a burning pain in the epigastrium soon after meals. His appetite becomes poor and capricious; often he may ask for sharply-seasoned foods, to pierce the numbness of his palate. It is said that there is a great distaste for meat, but the dislike is not specially noticeable in cancer as compared with other diseases. Under appropriate treatment the general condition of the patient may for a time improve; he may gain a few pounds in weight, and feel a better enjoyment of his food. The improvement, however, is transient only; there is soon a relapse, food is declined, or taken sparingly, vomiting commences, the body wastes, and the aspect of the patient changes almost daily for the worse. It is in patients such as these that especial care should be taken to make a routine examination of the stomach-contents. The presence or absence of free hydrochloric acid, the presence of lactic acid, and of Oppler-Boas bacilli should all be determined by a daily or two-daily examination. It is not sufficient to collect one sample of the stomach-contents for examination, a repeated analysis is necessary. The routine examination of the stomach-contents would, unquestionably, result in an earlier recognition, and therefore in a better and more successful treatment, than is possible at the present time.

In order to form some estimate as to the chances of life, of the condition of health, and of the relative values of different operations in patients afflicted with malignant disease of the stomach, it is desirable to enquire closely into the records of a number of cases, preferably in the practice of one surgeon, observed over a series of years. The fullest account of the surgical side of the question has been given by Krönlein, of Zurich, and by Von Mikulicz, of Breslau. The questions that are in urgent need of settlement are the following:—

1. Will a palliative operation upon the stomach prolong life?
2. Will it make the remnant of life more tolerable? Will it make the patient feel that the ordeal of operation is justified in the greater comfort of his later days?
3. Will a resection give a reasonable prospect of cure?

4. Will a resection, if followed by recurrence, give increased length of days and better health ?

It is necessary in fact to know whether, in the stage in which we now meet with the cases, an operation is worth doing, and if it is, whether it should be palliative, or whether an attempt should be made to eradicate the disease.

In order to have some means of comparing the cases surgically treated with those not operated upon, it is necessary that exact details should be kept of all cases coming to the surgeon, whatever their destiny, operation or internal medication, or absence of treatment, may chance to be.

Krönlein tabulates the cases which came under his observation from April 1, 1881, to February, 1902. All the cases were recorded without exception. There were 264 males, and the following table gives a brief sketch of their classification :—

A. Not operated upon :—		
1. Inoperable - - -	53	
2. Refusing operation -	14	
Total - -	67	
B. Operated upon :—		Deaths under Operation.
1. Exploratory laparotomies	73	7 = 9·5 per cent.
2. Gastro-enterostomies -	74	18 = 24·3 „
3. Gastrectomies - -	50	14 = 28 „
Total -	197	39 = 19·8 „

Of the 264 patients all but 13 were traced. Of these 13 the majority had not been treated by operation, and not one of them had been submitted to gastrectomy.

The fate of these patients is shown in the following table :—

Not traced - - - -		13
Dead—		
In consequence of operation -	39	229
Later, from intercurrent disease	2	
„ from suicide - -	1	
„ from the primary carcinoma - - -	166	
„ from recurrence after gastrectomy - -	21	

Living—		
Gastro-enterostomies	9	22
Gastrectomies	13	
		264

Of the 264 it will be seen that 67 were not operated upon (25·3 per cent.). In 53 of these an operation was contra-indicated; in 14 it was refused by the patients.

Of the 264, 73 had exploratory laparotomy performed (27·6 per cent.). After the surgeon had opened the abdomen and had become convinced of the impossibility of radical extirpation of the growth and of the unsuitability of a palliative operation, the abdominal wound was closed. Of the 73 patients, 7 died within the first week after operation (4 from exhaustion, 2 from pneumonia, and 1 from pulmonary embolism).

In 74 patients gastro-enterostomy was performed. At first sight this number appears small, but Krönlein has laid it down as a rule of his practice that in cases of carcinoma of the stomach where extirpation is impossible, the palliative operation of gastro-enterostomy shall not be performed unless there is evidence of stenosis. If this evidence fails, the operation becomes merely "exploratory." The only exception that has been made is in those cases in which there has been marked stagnation of food, without evidence of stenosis. Of the 74 gastro-enterostomies, 18 (or 24·3 per cent.) died under the operation.

Fifty patients were submitted to the operation of gastrectomy. Of these 14 died in direct consequence of the operation. Of the total 264 cases, therefore, only 18·9 per cent. underwent the operation. One of the 50 was the well-known case of total extirpation of the stomach, performed by Schlatter.

The subjoined table shows the percentage in which each form of operation was attempted:—

264 *Cases.*

No operation in		25·3 per cent.
Operation in—		
Exploratory	27·6	74·7 ”
Gastro-enterostomy	28·0	
Gastrectomy	18·9	

Operation-Mortality.

Exploratory laparotomy	9·5 per cent.
Gastro-enterostomy	24·3 „
Gastrectomy	28·0 „

In order to obtain a comparison between the life-chances of those patients who survive operation and those who are not operated upon, it is necessary to cancel the following :—

Patients who withdrew after examination	13
„ „ did not survive operation	39
„ „ died of intercurrent disease	2
„ „ committed suicide	1
	55

Deducting these 55 from the total of 264, we have 209 patients, who may be classified thus :—

A. Not operated upon :—		
1. Inoperable	51	
2. Declining operation	12	
	—	63
B. Operated on :—		
1. Exploratory laparotomy	58	
2. Gastro-enterostomy	54	
3. Gastrectomy	34	
	—	146
Total		209

The report as to these 209 cases at the beginning of March 1902, when these enquiries were concluded, was as follows :—

Died from carcinoma of the stomach	187
Living	22

There died :—

A. All those not operated on :—		
1. Inoperable cases	51	
2. Declining operation	12	
	—	63

B. Of those operated on :—

1. All the exploratory laparotomies	58	
2. Of the gastro-enterostomies -	45	
3. Of the gastrectomies - - -	21	
	—	124
Total of those operated on and not operated on -		187

There are still living :—

1. Of gastro-enterostomies - -	9	
2. Of gastrectomies - - -	13	
	—	22
		209

The duration of disease in patients suffering from carcinoma of the stomach was also reckoned. It was found that on the average the time from the first onset of symptoms up to the day of the patient's admission to the hospital, or up to the day of operation, was from eight to nine months. Means were also taken to discover the number of days from the patient's admission or from the date of operation to the patient's death.

The duration of time from entrance into hospital (time of operation) to date of death :—

A. In the non-operated cases - - -	102 days.
B. In the operated cases :—	
1. Exploratory laparotomies - -	114 days.
2. Gastro-enterostomies - - -	193 „
3. Gastrectomies - - -	520 „

If in these groups we sum up the values found for both periods of duration of the disease, we gather the following to be the average time for the whole duration of the carcinoma from the onset of the first symptoms till time of death :—

A. In the non-operated, 9 months and 102 days = about 12½ months.

B. In the operated cases :—
1. Exploratory laparotomies, 9 months and 114 days = 13 months.

2. Gastro-enterostomies, 9 months and 193 days = 15½ months.
3. Gastrectomies, 9 months and 520 days = 26½ months.

Krönlein, from this experience, draws the following conclusions :—

1. That carcinoma of the stomach, without operation, has a fatal termination in about a year.
2. That gastro-enterostomy prolongs the life of the patients suffering from this disease for about 3 months, on the average.
3. That gastrectomy, so far as it is followed by recurrence, prolongs life on an average about 14 months.

At the time this report was published there were 22 patients still living after operations. Of these 9 were cases of gastro-enterostomy and 13 of gastrectomy. The former will, of course, prove fatal within a few months. So far as the latter are concerned, their length of life since operation is shown in the following table :—

1 case is in the 8th year since gastrectomy.
1 " " 4th " "
2 cases are " 3rd " "
3 " " 2nd " "
6 " " 1st " "

Von Mikulicz in 1901 published the results of his experience from April 1891 to March 1901. During this period 458 cases of cancer of the stomach had been under observation in the Clinic at Breslau. This number includes 46 cases of cancer of the cardia. The diagnosis was confirmed either at the operation or by observation of the progress of the disease, or by examination after death. In several cases of cancer of the cardia, the diagnosis was confirmed by œsophagoscopy or by the removal and examination of a small portion of the growth. In 128 cases no operation was performed, chiefly because radical removal was no longer possible, and because there were no indications for any palliative procedure, such as gastro-enterostomy. In exceptional cases the operative interference was declined by the patient. The total duration of life from the commencement of the disease (as inferred from the symptoms in 67 cases in which it was possible to ascertain the facts) varied from a few months to 38 months, an average of $11\frac{1}{6}$ months.

In 320 cases recourse was had to operation as follows:—

1. Simple exploratory incision - -	44
2. Gastrostomy (in cancer of cardia) -	27
3. Jejunostomy - - - - - -	12
4. Gastro-enterostomy - - -	143
5. Resection of stomach - - -	100
6. Extirpation of stomach - -	3

Exploratory Incision.—Four deaths followed this operation, giving a mortality of 9 per cent. The average duration of life after operation was $4\frac{8}{10}$ months, and from the beginning of the disease $14\frac{3}{10}$ months. If the cases submitted to exploratory laparotomy be added to those in which no operation was performed, we get an average duration of life of a little over 13 months, from the beginning of the disease, a figure which is adopted by the author as a basis for judging of the results of the operative treatment of gastric cancer.

Gastro-enterostomy.—There were 48 deaths in 143 cases, a mortality of $33\frac{1}{10}$ per cent. Although during the last three years the mortality has been reduced to 26½ per cent., it is still very high in proportion to the same operation in non-cancerous conditions. The average duration of life after operation was $6\frac{4}{10}$ months, and from the beginning of the disease 14 months; but if the operation deaths are included this is reduced to $12\frac{3}{10}$ months, slightly shorter than the duration of life when no operation is performed.

Resection of the Stomach.—There were 37 deaths in 100 cases. In the last three years the death-rate has been reduced to 25 per cent., that is to say, a mortality slightly lower than that of gastro-enterostomy. The results, as observed in 58 patients who survived the operation, and whose subsequent history is known, are as follows:—

Twenty are still alive between six months and eight-and-a-quarter years.

Longer than	1 year	17	are alive.
„ „	2 years	10	„
„ „	3½ years	4	„

These last four may be regarded as radically cured. The author regards these results as quite as good as those after operations for cancer of the tongue or rectum.

It will thus be seen that there is no great divergence in the results taken from the two clinics, allowing for the fact that the number of cases in the one is almost double that in the other, while the period covered by the observations is only one-half. Krönlein records 264 cases, extending over 21 years, Von Mikulicz 458 in 10 years. The results may be compared in this way:—

	Krönlein.	**Mikulicz.**
The non-operated cases lived from the beginning of the disease - - - -	12½ months.	11⅙ months.
The cases treated by exploratory laparotomy - -	13 „	14$\frac{3}{10}$ „
The cases treated by gastro-enterostomy - -	15½ „	14 „
The cases treated by gastrectomy - - - -	26½ „	24½ „

A careful study of the wealth of experience laid bare in these records will enable us more clearly to formulate our ideas as to the principles that should guide us in dealing with this most serious disease. In the first place it must be admitted that our means of obtaining cases sufficiently early are almost as meagre as it is possible for them to be. Until more accurate methods of diagnosis are established, it is absolutely imperative that recourse should be had earlier and more often to the exploratory laparotomy. At present that operation is limited to the examination of patients when the diagnosis has been made, and when the only question to be settled is whether the growth is removable or not. But in order to better our results we must explore, not to *confirm*, but to *make*, a diagnosis.

When the diagnosis has been made, and the patient is submitted to operation, it is difficult to decide upon the exact surgical procedure which it is wisest to adopt. Von Mikulicz, Krönlein, and not a few other surgeons have spoken strongly upon the question of gastro-enterostomy, saying that this method should be adopted only in cases where stenosis, either at or near the pylorus, is caused, or in cases where stasis of food is marked. There can be no doubt that in such cases gastro-enterostomy is productive of the most remarkable benefit to the health and well-being of the patient. The weight increases, the appetite and the power of gratifying it return, and vomiting, often the most distressing and unceasing symptom, stops at once. But there can also be no doubt that in some instances, when the growth does not actually obstruct by its bulk the onward passage of food, a decided benefit results from the operation. The stomach is better and more quickly drained, and, as a rule, food can be taken more frequently and with greater comfort. In the majority of these cases, however, little or no benefit results from gastro-enterostomy. The position, therefore, may be thus briefly stated. If, after exploration, a growth is found to be obstructing the pylorus, or to be narrowing the stomach and causing an hour-glass condition, or if stasis of food has been a marked symptom, then gastro-enterostomy will give very decided relief. If, on the other hand, the growth be confined to one or other of the curvatures, and if neither stenosis nor stasis be present, gastro-enterostomy will give little or no relief; it will not prolong life nor give a greater degree of comfort.

The operation of gastrectomy in the hands Von Mikulicz and of Carle and Fantino has given a less mortality than gastro-enterostomy. Its advantages as compared with gastro-enterostomy are that it gives a greater prolongation of life—ten or eleven months longer—and that it affords a greater degree of comfort to the patient. Though recurrence may follow, yet in the majority of cases the patient is relieved greatly by the removal of a foul ulcerating growth, from the surface of which an offensive and septic discharge is constantly occurring, and from which hæmorrhages, more or less copious, are often likely to take place. If after removal of a malignant growth of the stomach the surgeon will open the viscus and inspect the surface of the tumour, he will realise, when he sees the foul, ulcerous mass, that its removal cannot but be of vast benefit to the patient. It is true that in the great majority of cases the growth will recur either locally or generally, but the question may arise as to whether gastrectomy should not be performed deliberately as a palliative operation even in cases where an early secondary deposit can be seen in the liver, or inaccessible or irremovable glands be found in the pancreas or along the aorta and vena cava. If we take into account the following advantages of gastrectomy as compared with gastro-enterostomy—that in the most competent hands its mortality is not greater, but is even less, than the mortality of gastro-enterostomy; that a prolongation of life for ten months longer than the period given by gastro-enterostomy is the rule; that the comfort, the general health, the appetite, and the well-being of the patient are all emphatically better; and, finally, that the patient has always a chance, even though it is of the slenderest, of a complete recovery from his disease—if we take all these into our consideration, there can be no doubt that the operation of choice will always be gastrectomy. Gastrectomy will be done always when a radical operation is attempted; it will probably be done often when nothing more than a palliative operation is intended.

If, after the abdomen has been opened, it is found impossible or imprudent to attempt either gastro-enterostomy or gastrectomy, some relief may be obtained by performing duodenostomy or jejunostomy. The scope of these procedures is very strictly limited, but in rare instances, when the prolongation of life for even two or three weeks is of the greatest importance, then either of these operations can be done with propriety. The operations are simple, speedily done, and cause little or no shock, and they can, if need be, be readily performed with cocaine-anæsthesia.

REMARKS ON SOME RESULTS OF OPEN-AIR TREATMENT.[1]

By CLAUDE C. CHIDELL, M.B., B.S.,

Medical Superintendent of the London Open-Air Sanatorium.

GENTLEMEN,

VERY much has been said about methods of open-air treatment—the open window, the value of climate, of diet, and of forced feeding, the proportions of rest and exercise—all of which has its value in assisting the public, both medical and lay, to learn the principles of so-called "natural" treatment. Much, too, has been said about the results of this treatment, and some promises have been made which we shall never see fulfilled. The vast experience of systematised treatment which has been accumulated in Germany and analysed by the Imperial Health Office has led to valuable conclusions which those who can may read, but a humble note on our small observations at Pinewood is all the original material that I am able to offer you to-day. Pinewood is yet too young to afford material for deducing ultimate results, and I shall only attempt to derive a few preliminary conclusions from watching 300 cases here, and then pass to the matter of statistical results.

EFFECT ON SYMPTOMS.

Let me take first the effect of open-air treatment on a few typical symptoms. One may say that in nearly all but the advanced cases which are nearing their end the symptoms tend to improve.

1. Flesh in most cases is gained, rapidly at first, then more slowly, and the muscles become firm as exercise increases. The improved muscular tone gives a better carriage to the stooping shoulders, and the gait, which may have been weak and fearsome, becomes confident and manly.

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[1] A paper read before the Home Counties Branch of the Incorporated Society of Medical Officers of Health.

2. Colour returns to the pale cheeks, and the wearied and wan look gives place to a healthiness of appearance far beyond that of even most country dwellers. This improved colour is due partly to increase of hæmoglobin and partly to sunburn. It is worth noticing that it was currently said in Davos that the sunburn did not occur in some of the worst cases, and I think the observation is a true one and applies also here.

3. Cough almost always decreases—firstly, because the patient, if not too stupid or obstinate, soon learns the advantage of suppressing useless cough; secondly, because foul air and over-exercise, such as fighting against a wind, are avoided; and thirdly, because the sputum often diminishes in amount.

4. As might, therefore, be expected, expectoration does not decrease in the same ratio as the cough, and indeed seldom absolutely disappears in three or six months' treatment, except in very slight cases. The decrease one hopes to see is progressive from month to month, for sudden variations occur, with or without obvious cause, and a sudden, almost complete cessation of expectoration seems to be one of the symptoms of that fatal complication, tubercular meningitis.

In general, however, one may say that the existence of sputum definitely coughed up is one of the most important symptoms of organic disease of the respiratory tract, and that in tuberculosis increase or decrease of sputum is a valuable sign of waxing or waning disease. Tuberculosis is usually a chronic disease, and expectoration once established is slow to disappear; and though it may persist long after all active disease has subsided, its presence should always raise suspicion and lead to investigation. On the other hand it is, strange to say, absent in some cases of extensive disease in which the physical signs would lead one to expect it.

5. Night-sweats have very commonly been present in our cases previous to admission, and they sometimes continue for a few days, but only in one or two among 300 cases observed at Pinewood have they persisted in anything like the same intensity. This disappearance of night-sweats has been observed in many sanatoria and hospitals worked on open-air lines, and to me it seems the most striking result of open-air treatment, because it occurs almost suddenly and almost always.

Without wishing to disparage the proper use of drugs, I may add that such as we have used when open-air has partly or entirely failed have not succeeded in any marked degree; and it is evident that the first essential in dealing with night-sweats (and with the disease of which they are a symptom) is a constant and thorough flushing with pure air by cross-ventilation. No drug can be a substitute for this simplest and most certain remedy when combined with complete rest. Rest alone does not produce the desired effect which, further, comes on too quickly to allow one to suppose that mere increase of food is the cause, so that the abolition of this distressing symptom seems to be attributable directly to open air.

Without going further it is safe to say that the results obtained in symptomatic improvement amply prove the soundness of the principles of the treatment quite apart from statistical evidence—just as I presume most of you would say that direct clinical observation suffices to prove the value of diphtherial antitoxine.

EFFECT ON PHYSICAL SIGNS.

The second point to which I wish to invite your attention is the result of treatment on physical signs, and I will confine my remarks to changes in râles, because I take these to be usually the most indicative sign of increasing or diminishing disease.

We should bear in mind how coarse are our best means of gauging the intensity and extent of lung-disease. It is certain that the stethoscope fails to detect disease in its earliest stage, and post-mortem evidence always shows the extent to have been greater than the signs indicated, so that there is no doubt that any change must be relatively considerable before it is evidenced by physical signs. We cannot, therefore, be surprised if we fail to detect a diminution of râles in such a slow-paced process as tuberculosis, even though we feel certain from the lessening of expectoration and other symptoms that improvement is really taking place; and it becomes necessary to distinguish between actual betterment of local disease and the physical signs of such improvement. It is fair, then, to infer local improvement in a given case from the symptoms even when a careful examination detects no diminution of râles.

In our experience at Pinewood it is common to observe a marked improvement of both general and local symptoms in three to six months' treatment, while the physical signs are found to have scarcely altered. The greatest improvement as regards râles has been observed in the younger patients, and in cases where the râles were believed to be pleural.

Though one may feel disappointed that râles are often so slow in disappearing, it must be remembered that they do not necessarily argue activity of disease, and that many patients may keep their râles and their health too, while hard at work. Very chronic cases of unfavourable tendency may show little change in signs and are to be judged rather from their symptoms; but cases with more acute disease usually give clear evidence of spread of diseased area in the course of weeks or months.

The recognition of the slowness of the healing process has led numerous workers to try and hurry it on by the use of tuberculin, and some have claimed that they obtain a more rapid result by the use of this remedy, while others speak with reserve and only claim that no harm ensues. Our experience at Pinewood is far too small for generalising, and one can only say that in three slight cases treated for 10, 15, and 17 weeks and receiving maximal doses of 4, 60, and 225 mg. of the old tuberculin respectively, the signs and symptoms did not in the long run alter more than in others not so treated. A physiological tolerance of the drug was established, but whether these patients are less liable to recurrence than others cannot yet be judged. All the patients improved, but the effect of the tuberculin remains ambiguous.[1]

STATISTICS.

Statistics are in many cases based on the combined results both in symptoms and in physical signs, and since general improvement is so common, it is no wonder that a large percentage of cases are returned as "improved." Some sanatoria give figures showing proportions of "cures," but I protest against the use of this term, as applied to patients just after a course of treatment, seeing that relapse is notoriously common in pulmonary tuberculosis; and it is a remarkable fact that in some cases the great improvement of the first weeks or months in a sanatorium is gradually lost.

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[1] I am indebted to Drs. Hillier and Hector Mackenzie for permission to publish these cases.

Why it should be possible to stem the tide when it is fast ebbing, yet impossible to hold the gain when the tide seems to have turned, is hard to say, but this may sometimes be noticed even under the ideal conditions of a sanatorium. Under the relatively unfavourable conditions to which many patients return, ultimate relapses are very numerous among the advanced cases which form the bulk of those sent into English sanatoria. Looking back on nine years' association with large numbers of consumptives in Swiss, German, South African and English health-resorts, I can only give my impression that the mortality is very large, and I may add that I include Nordrach in the foregoing remark. Yet it has been claimed that Nordrach produces 90 to 95 per cent. of cures in all stages—one of the most unwarrantable assertions ever made, and not, I believe, supported by the authority of Dr. Walther, who admits even the most advanced cases.

At many sanatoria the total improvements amount to 70 to 80 per cent. These include, of course, many cases temporarily benefited, and only show that open-air treatment has an extraordinarily favourable effect on the life-processes in consumptives, but are no index of arrest of disease, much less of cure. Apart from irresponsible statements, it must be remembered that in these matters the personal equation counts for much. Terms require definition, and it appears to me that the best result obtainable after several months' treatment merits only the designation "apparent arrest," by which is meant that the patient is fit for work, and that fever and all symptoms of *active* disease are wanting. Râles, sputum or tubercle-bacilli would not necessarily prevent a case from being regarded as arrested, but the râles must be at least stationary over a period of several months, the sputum small in amount, and bacilli inconstant or at any rate few in number. "Fitness for work" means that the patient can return to his former employment, or do the work of a healthy man in some other.

The word "cure" may be arbitrarily, but conveniently, defined as a condition of arrest maintained for two years, while "absolute cure," like happiness, can be predicated of a man only when he is dead, or possibly during life by the diagnostic use of tuberculin.

Further, the stages of the disease must be clearly defined, and a common classification agreed upon, so that the results of numerous sanatoria may be available for comparison. I do not propose to discuss the merits of the various systems of classifying cases of pulmonary tuberculosis, but these ought to be the subject of a representative conference of interested parties, convened to adopt some one system for this country.

There would be obvious advantages in adopting the system of the German Imperial Health Office, which is based on a consideration of both the intensity and the extent of the lesion, though it seems more open to objection than Dr. Turban's system.[1]

Percentages of results have but little value unless they are related to the stages of the disease, and one must expect the British results to be less satisfactory than contemporary German figures, because there can be no doubt that proportionally far more "third-stagers" find their way into our sanatoria. If the public sanatoria and hospitals of this country are to be used to the best advantage, it is essential that no patient should be retained simply because he has a subscriber's letter, but the medical staff should have a free hand in selecting suitable cases. Then, as time proceeds, no doubt medical men will send in a larger proportion of those early cases which alone repay treatment in an economic sense, for it may well be questioned whether it be wise in all cases and at all costs to prolong life when there is no hope of self-support, while others who might be saved are drifting into the same wretchedness for want of early diagnosis and treatment.

The vital questions as regards results are to be answered in terms of capacity for work and duration of working life. The question "what proportion of patients leave sanatoria capable of earning their living, or with an early prospect of so doing?" has hitherto received but partial answer in Britain, owing to the fact that the cases are not often classed in stages; and the second question "what proportion of patients keep well?" can as yet receive no answer on a sufficiently large scale, because it is but yesterday that numerous sanatoria came into being in this country.

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[1] Dr. Turban's stages are as follows:—

I. Slight affection of not more than one lobe or two half-lobes.

II. Slight affection of not more than two lobes, or severe affection of not more than one lobe.

III. All beyond II.

See further, article by Dr. Camac Wilkinson, *Brit. Med. Journ.*, June 7, 1902.

I will not give any Pinewood figures relating to the first question because we are still too immature to deal satisfactorily with them, but I will quote, as a most instructive example, a table of permanent results dealing with 541 cases treated by Dr. Rumpf at Friedrichsheim in the year 1900, which is brought up to date with the latest information—in other words, the report extends to two to three years after discharge.

PERCENTAGES.

Stage.	Able to work.	Unable to work or receiving aid.	Dead.	Treatment repeated.	Unknown.	Total.
I.	75·6	6·8	3·4	11·5	2·7	= 100
II.	62·4	9·2	12·8	12·8	2·8	= 100
III.	25·4	14·7	54·8	4·0	1·1	= 100

NOTE.—The results are really more favourable than the figures show, because when treatment was repeated there was often no relapse; because, of the "unknown" cases, many may be able to work; and because several of the "dead" died from other causes (one of suicide).

Finally, gentlemen, I appeal to your influential Society to help in rendering two great services to the cause, firstly, by insisting that a strict selection, based on physical condition, shall be made of applicants for admission to public sanatoria; and secondly, by aiming at the adoption of some basis of classification which shall serve as a common ground for the elucidation of results and dispel the present confusion of tongues.

Postscript.—Since reading the above paper I have seen the Report of the First International Tuberculosis Conference. The question of the classification of cases of pulmonary tuberculosis was there discussed. It would be a great gain if the matter could be settled internationally.

IMMUNITY AND TREATMENT.

By ALBERT S. GRÜNBAUM, M.A., M.D., F.R.C.P.,

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THE number of experimental data available for the elucidation of the various theories of immunity is now so large that the comprehension of the terminology of these theories—which, once understood, makes their description a comparatively easy matter—may be best attained by the consideration of some of the fundamental experiments.

It is nearly useless to attempt to define immunity. Like many other words the term immunity has an intuitive meaning conveyed by a knowledge of its phenomena, and it is at present impossible to give a definition in such general and logical terms as to embrace all that is meant by and included in the word. For our purposes we may regard immunity as the power of the organism to preserve itself against present or future destructive attacks, whether endogenous or exogenous, and whether the opponent be vitalised or merely organised—in other words, its power of self-preservation amidst variation of environment. It appears at once from this definition that no organism can be completely immune or is likely to be utterly unprotected.

In ordinary parlance when we speak of an individual as immune to any given infectious disease, *e.g.*, scarlet fever, we usually imply either that he has been exposed to the disease sufficiently often to demonstrate his immunity, or that, having passed through an attack, he is likely to remain exempt in spite of future exposure to infection. But the immunity in these two instances is not necessarily the same, and we may say that in the first instance the individual was possessed of *congenital*, and in the second of *acquired*, immunity—while in each case the immunity is obviously *natural*.

As a contrast to these we may take the cases of a horse which has been injected with diphtheria-toxine for the purpose of producing anti-diphtheritic serum, and an individual who, having been exposed to the infection of diphtheria, has been given a prophylactic injection of the horse's serum. In both instances the individuals should be immune. But the horse has obtained its immunity as the result of its own resistance to the diphtheria-toxine—it possesses an *active* immunity; whilst the person who has received the prophylactic injection of anti-toxine has not really himself contributed towards his immunity, which is therefore termed *passive.* But in both these instances the immunity produced is *artificial.* We shall see later that these distinctions are of practical importance.

For the production of acquired immunity an antecedent infection is generally necessary. It is obvious that the details of this process must be subject to considerable variation, with corresponding variation in the resulting immunity. Thus, the action of the tubercle-bacillus is largely mechanical and it produces fibroid and degenerative changes in the tissues which it attacks. In contrast to this, the diphtheria-bacillus remains at the site of inoculation, and there prepares the toxine which produces the symptoms of the disease. Yet other bacteria are capable of multiplying in the tissue as well as of producing poisons, and thus exert their influence in both ways. Infection can occur, as a rule, only upon special soil. Thus, while the comma-bacillus in the intestine produces cholera, it will cause but little harm and be rapidly destroyed, if introduced beneath the skin; while conversely the anthrax-bacillus beneath the skin produces severe illness, but taken into the alimentary canal is nearly harmless. The tubercle-bacillus, on the other hand, can enter, settle, and multiply practically anywhere. But infection implies more than the mere presence of the bacillus. It must be remembered that the tubercle-bacillus has been found in the sputum, the diphtheria-bacillus in the throat, and the typhoid-bacillus in the stools of healthy persons. Nor does the presence of such bacteria necessarily imply immunity of the individuals concerned. The two terms are merely relative; there is no certain infection or absolute immunity.

There seems to be a general underlying principle, of which the production of immunity is merely a special instance; namely, the resistance of the organism to the introduction of any foreign substance, with a consequent production of corresponding antagonistic substances. For example, the injection of egg-albumen—either intraperitoneally or subcutaneously—into the rabbit leads to the formation in the rabbit's blood of a substance which when added to a solution of egg-albumen precipitates the latter. This formation of *antidotes* is not, therefore, limited to poisonous bodies; it occurs with most foreign colloid substances. The antidotes are not formed against crystalloids, whether simple organic salts or complex alkaloids.

Upon the fact just described, and upon the principle enunciated by Weigert that repair overcompensates the injury, depends the preparation of antitoxic and other curative sera. The injected toxine stimulates the organism to produce more than the corresponding neutralising quantity of antitoxine.

For the intelligent and efficient administration of curative sera some conception of the mechanism of infection and immunity is required. Various hypotheses have at different times been put forward, each probably containing some elements of truth. Thus, while Metchnikoff embraces a vitalistic view, Ehrlich adopts a more purely chemical explanation, while Zagger has lately put forward a physical hypothesis. Inasmuch as physiological or pathological activity is the result of chemical changes which themselves depend on variations in physical conditions, the divergence between the various theories may not be so great as would at first sight appear.

By far the greatest amount of work and attention has been bestowed on the chemical view—Ehrlich's "side-chain theory" —both by Ehrlich and by others, so that it may be taken as the basis for our description, while the other theories will be incidentally brought in when necessary. For the object of this article is not so much to present the theoretical aspects of the phenomena of immunity as to give the foundations upon which practical treatment depends.[1]

Although it would be more logical first to examine the phenomena of infection, it will be easier to start with a consideration of the action of toxine and antitoxine in the case of diphtheria.

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[1] This is not the place to enter upon a discussion either of the tenability of Ehrlich's theory or of the criticisms levelled against it. Of the latter, those by Gruber and others have often been much to the point, and have led to the investigation of many interesting questions But I think that Ehrlich's critics have so far succeeded only in showing that he may not have offered a strict proof of some minor hypotheses; they have not succeeded in showing him to be

The action between these two takes place according to the ordinary chemical laws of combination. That is to say, the toxine and antitoxine can be mixed in such proportions in a test-tube as to produce a mixture which, injected into an animal, will have no pathogenic effects ; just as dilute solutions of hydrochloric acid (HCl) and bicarbonate of soda ($NaHCO_3$) might be mixed in such proportions as to produce a normal saline solution with no pathological properties when injected, although the acid by itself would cause destruction of tissue.

Moreover, we should find that on doubling the quantity of toxine (provided the initial quantity were large enough) we should require practically twice the quantity of antitoxine for its neutralisation. From this it is clear that the more severe the infection, the greater the quantity of antitoxine which must be administered to effect a cure.

It was found, both by experiment and by experience, that time was an important factor, and that the longer the interval since infection, the greater the quantity of antitoxine required. While in the case of human infection this observation might be explained by the greater quantity of toxine produced by the bacteria in the longer period, such a reason was obviously inapplicable to the case of animals injected with a definite amount of toxine without any bacteria.

It is known that the various toxines have different predilections for various cells of the body, *e.g.*, the diphtheria-toxine for nerve-fibres, tetano-toxine for the motor-cells, &c., so that, pursuant to the chemical analogy pointed out above, it is not unnatural to suppose that the toxine enters into combination with the cell, from which it can be released only by an increased mass-action, *i.e.*, by an excess of antitoxine. The longer and closer the combination of cell and toxine, the larger the quantity of antitoxine necessary to dissociate them. It follows also that if the affinities between cell and toxine, and

wrong. For the rest, the differences are largely on questions of terminology. I consider such terms as toxone, toxoid, &c., to be useful and graphic, even if they represented nothing more than the physiological effect of certain neutralisation-percentages of toxine and antitoxine; and Madsen himself, who, as well as Gruber, has attempted to disprove their existence, retains the term *toxons* on account of its convenience.

Consequently, for the purpose of this paper, I adopt Ehrlich's theory, and to a large extent his nomenclature, as giving the most graphic explanation of many known facts, and because it prophetically led to the discovery of many of them.

between antitoxine and toxine, are only slightly different, it may be impossible to effect the dissociation.

This union of the toxine with the cell is not merely a hypothesis, for we find that when an animal is immune to a particular poison, this poison may be found, after injection, in the circulation for some considerable time ; or it may be united to some cell on which it has no action, *e.g.*, tetano-toxine to the liver-cells of the scorpion. But in a susceptible animal the greatest quantity of toxine is found in the affected cell (*e.g.*, tetano-toxine in the brain-cells of the rabbit), and may be extracted from it.

Two hypothetical deductions were made by Ehrlich from these and other facts : (1) that the molecular group by which the toxine unites with either cell or antitoxine is the same, since it cannot simultaneously unite with both ; and (2) consequently the antitoxine is actually the very cell-constituent with which the toxine unites, cast off from the cell and free in the circulation. The molecular group in the cell to which the toxine becomes attached was termed by Ehrlich the *receptor*, and antitoxine consists of free receptors. The molecular group in the toxine which unites with the receptor he termed the *haptophore* group (ἅπτω, I bind). Since diphtheria-toxine may by time be so modified as to lose its toxic power whilst retaining its neutralising power for antitoxine, Ehrlich assumes also the existence of a molecular group exerting the specific toxic effect, and this he calls the *toxophore* group. These conceptions serve to explain many phenomena.

The modification of toxine is called *toxone*. From what has just been said concerning its diminished or absent toxicity with remaining power of neutralising antitoxine, it is natural to assume that the toxone has the same *haptophore*, but not the same, or no, *toxophore* group. We might roughly compare toxine and toxone to sodium hydrate (NaOH) and sodium bicarbonate ($NaHCO_3$) respectively : both can neutralise hydrochloric acid, although the latter has practically no alkaline reäction.[1]

But the reäctions occurring in processes of immunity are not always so comparatively simple as between toxine and antitoxine. In bactericidal sera there are at least two bodies concerned in producing the effect. This is proved for many phenomena by the fact that heating "immune" serum (viz., the serum obtained from an immunised animal) to 56° C. destroys its action; but it can be *reäctivated* by the addition of some normal serum, although normal serum itself is inactive. Moreover, if the normal serum be heated to 56° C., it loses its reäctivating power. Consequently, this heat-sensitive substance is probably the same in both normal and immune sera, and is not the result of the immunising process; whilst the other bodies necessary for the bactericidal action exist only in the immune serum.

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[1] The recent attempts to demonstrate the non-existence of toxones have been alluded to in the previous footnote. It would lead too far to discuss them. The results are important and interesting, as might indeed be assumed, since they come from such observers as Arrhenius and Madsen, and Gruber.

It will be shown immediately that the substance produced in the serum of immunised animals unites on the one hand with the cell to be destroyed and on the other with the substance (present in normal serum) required to complete the reäction. The first substance is thus appropriately termed the *mediator*, since it links the cell with the other substance, or the *amboceptor*, uniting as it does with both; while the second substance whose presence is necessary for the completion of the reäction is termed the *complement*.

Experimentally the presence of two bodies taking part in the reäction can be most easily shown with the blood-laking or hæmolysing bodies produced in the serum of an animal by the injection into it of the blood-corpuscles of another kind of animal. The method of experiment is as follows:—The serum of a goat immunised against sheep's blood is heated to 56° C. to destroy its hæmolytic power (by the destruction of the complement). To this heated immune serum are added sheep's xanthocytes (red corpuscles), and after standing awhile, they are again separated by centrifugalising. If the mediator has been abstracted by the xanthocytes, then, on addition of normal serum to the centrifugalised xanthocytes, hæmolysis ought to occur. This does actually take place. The xanthocytes have therefore acted like the toxines and neutralised the mediators, which correspond to the antitoxine, in the immune serum. On the other hand, if the mediator has thus been removed from the immune serum, then on adding fresh normal serum (containing complement) and fresh xanthocytes (to be hæmolysed) no hæmolysis should occur. Nor does it. By suitable variation of the experiment it can be shown that the complement will not unite directly with the xanthocytes.

Having thus briefly outlined some of the phenomena and theoretical foundations of immunity we may proceed to examine a few of the practical applications.

Prophylactic inoculation.—Applied to smallpox this method of immunisation is well known. The reäction produced is generally considered to be a modified form of cowpox, itself a modification of smallpox. I think a better explanation is given by the hypothesis that vaccine contains the toxine of variola in a "toxone" condition. It thus immunises without producing the toxic effects of variola in a similar way to that in which diphtheria-toxone leads to immunity. A certain interval elapses before protection is established, and as a result vaccination is ineffectual against smallpox, if done later than four days after infection. It seems to be a disputed point whether vaccination then favourably or unfavourably affects the course of the disease: we shall consider this point immediately.

It is obvious that prophylactic inoculation may, in accordance with theory, produce a temporarily reduced resistance, and consequently increased liability to infection. For if the cell-receptors are partly occupied by the haptophore group of the vaccine, and then, before sufficient time for their hypertrophic reproduction has elapsed, the virus seizes on the remaining receptors, the chances for reproduction are diminished, and the cell worse off than if no vaccine had been introduced. It is consequently, as a rule, important to avoid infection during this period of diminished resistance, so that inoculation against enterica should be done some weeks before the individual is likely to be exposed.

But the conditions are not quite the same when infection has already taken place. In such cases subsequent inoculation may be a successful form of treatment. Thus, a dilute anti-typhoid *vaccine* has been used in enterica, and the utility of tuberculin in the rational treatment of tuberculosis is well known, although perhaps not so generally known as it ought to be. The explanation of these results is probably that other tissues than those specially affected by the toxine take part in the formation of antidotes.

At first sight one might think that the introduction of the toxic tuberculin into a consumptive patient, who is already manufacturing toxine in his own body, was likely to do him more harm than good. But, as a matter of fact, this is not so, especially in cases of simple infection. For the autochthonous toxine is formed at, and its action chiefly limited to, an area of cells which are obviously too weak to resist, whereas the other tissues may be and are stimulated by extraneous toxine to produce suitable antidotes.

In accord with the opinion and experience of others, I think the tuberculin-cure has been unfairly discredited, and the sanatorium treatment of phthisis unduly extolled. The two together form the ideal treatment. (The combination of suitable therapeutic, climatic, and nutritional conditions is not the monopoly of the treatment of phthisis, but of all disease.) Sanatorium treatment can at most induce a general resistance: it does not produce any specific improvement, as the subsequent tendency to relapse shows. The diathetic deficiency in the special mediators antagonistic to the tubercle-bacillus can be overcome only by the stimulus to their production effected by the tuberculin.

An attack of the disease is often the most effective and sometimes the only way of producing immunity. At present there is no other method by which immunity to many infectious diseases, *e.g.*, measles, scarlet fever or typhus, may be conferred. Yet in some cases of bacterial infection, even if the the individual survive, there may be little evidence of acquired immunity. In man, after recovery from tetanus, there is but little, if any, antitoxine in the blood. From what has gone before it will be clear that a mild attack will probably produce greater immunity than a severe one, for in the latter case only a sufficient quantity of antidote may be formed, and no excess.

The protection conferred by an attack of the disease or by a process of active immunisation (vaccination) is always superior in permanence, and usually in amount, to that attained by the injection of an antitoxic or bactericidal serum obtained from an immunised animal. The reason seems to be that the protective bodies formed in the immunised animals are more or less specific. In the case of antitoxic sera the difference is not so marked, and the diphtheria-antitoxine obtained from the horse seems to answer well enough to neutralise the diphtheria-toxine in man. But the attempt to immunise a man by repeated injections of horse-antitoxine would probably result in the formation of an anti-(horse)antitoxine, and produce the reverse of the desired effect. The difference is more marked in the case of bactericidal sera. Thus the mediators formed by the goat against the typhoid-bacillus are neither the same, nor so efficient, as those formed in man, for man. Moreover, a suitable complement is necessary for effective action, and no complement is so suitable as that of the kind of animal by which the mediator has been formed. Within limits, different complements may be able to complete the reäction, just as several different keys may fit one lock, although one particular key does so best.

In any case, the quantity of immunity will be merely that contained in the introduced serum, which itself is so far from being a stimulus for the production of fresh mediators that it leads rather to the formation of anti-mediators. Since then the anti-typhoid mediators of the goat, for example, are specific, and their introduction into another animal, *e.g.*, man, would lead to the production of an anti-anti(goat)typhoid mediator, but not of an anti-anti(horse)typhoid mediator, it seems probable that any typhoid-immune serum contains merely an increased number of the mediators normally present, and not new ones specially formed, for the typhoid-bacillus—for then the serum ought to be equally effective in all animals. It also follows that the mediators exist separately for each bacterial species.

The short duration of passive immunity is thus seen to be due to the introduction of an alien immune serum, leading to the formation of anti-antidotes which neutralise and render useless the mediators introduced with the immune serum. Further, the long duration of active immunity is explained by the stimulated production of mediators already present, and to these, of course, no anti-mediators will be formed.

But the administration of immune sera for curative purposes also requires some care and must not be done in a mechanical and routine fashion. Antitoxic sera will not, it is true, be generally given in excess; and too much anti-toxine is certainly better than too little. Since the action is subject to chemical laws, we must remember that the longer the illness before the injection of the serum, the more firmly will the toxine be united with the cell and the greater will be the quantity of anti-toxine necessary for the dissociation of toxine and cell.

The administration of bactericidal sera is not so simple. If more mediators are introduced than there are complements to complete them, the complements may partly or entirely become attached to free mediators, and only a few or none to those attached to the bacteria; consequently the bactericidal effect will remain uncompleted. Although no strict comparison, this conception may be aided by imagining an attempt to cure a case of poisoning with corrosive sublimate by the introduction of exactly sufficient potassium iodide to precipitate all the mercury salt. An excess of potassium iodide would keep the salt in solution and enable it still to exert its poisonous effect (assuming for the moment that HgI_2 and $HgCl_2$ are equally poisonous). The administration of bactericidal sera consequently requires care, and the especial point to be observed is the ensuring of the presence—and therefore, if necessary, the introduction—of sufficient complement. Sometimes the human complement itself may be suitable for completing the introduced serum, but it should be remembered that disease and other causes are liable to diminish the quantity. Towards the end of fatal illnesses the serum-complement is often much lessened—a fact which accounts for terminal infections. Alcohol has a similar effect. As yet practical serum-therapy has not satisfactorily overcome this difficulty of ensuring the presence of suitable complements.

So far we have considered only bacteria and their toxines. But in accordance with the principle enunciated above we should anticipate the possibility of the formation of antidotes to cells. In many cases they can be produced; *e.g.*, the injection of the liver-cells of a guinea-pig into the peritoneal cavity of a rabbit leads to the formation of a substance in the rabbit's blood, such that the rabbit's serum injected into a guinea-pig produces a condition analogous to acute yellow atrophy of the liver. In other words, the serum of an animal which has received injections of particular tissue-cells derived from another species contains a substance capable of destroying similar cells within the body of a living individual of the second species.

Organotherapy as a mode of treatment is yet in its infancy, but at least one great success has been achieved in the treatment of myxœdema. For while there may be some doubt as to whether atrophy of the thyroid is the cause or the result of myxœdema, there can be no doubt that its presence prevents, and its administration cures, the symptoms of the disease.

It is a distinctive feature of organotherapy or cellular therapeutics[1] that the remedies can be administered by the mouth, that they are not destroyed by digestion, and can be absorbed from the alimentary canal. This holds good, not only for the thyroid gland, but also for the anti-thyroid serum given for the cure of exophthalmic goitre. This serum consists simply of the serum of sheep whose thyroids have been removed about six weeks previously.

The explanation of the therapeutic action of thyroid gland and of antithyroid serum is not quite easy. But it is quite in accordance with Ehrlich's theory that the substance produced in the absence of the thyroid should neutralise that produced by an excess of thyroid, and I have introduced this digression rather to call attention to an aspect of the subject well worthy of development along the lines and on the principles of serum-therapy.

Finally, it may be well to devote a few words to the question of predisposition and susceptibility; for preventive is better than curative medicine. Of the existence of a condition of diminished resistance expressed by these two words there can be no doubt. Applying, as Ehrlich has done, the theory to general biological questions, we assume that some of the cell-receptors are used for nutritional purposes. But when they are not occupied by a nutritive molecule, they present a point for attack by some toxine-molecules, and as a natural consequence the badly-nourished organism will be more liable to disease. For the same reason the popular notion of greater liability to infection during hunger is very probably correct: some receptors are then unoccupied. Moreover the nutritive molecule must have such a constitution as to possess greater affinity for the receptor than the toxine-molecule, or the latter will be the victor. Susceptibility may also be increased, as we have seen, by a diminution in the amount of complement; and it is quite possible for this deficiency of complement to be congenital and consequently hereditary. At present we do not know of any other means of increasing the complement than by inducing a good state of nutrition. This must be our first line of defence; its importance cannot be overrated, and yet is mostly under-estimated.

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[1] In a few cases other sera have been successfully given by the mouth, and when the administration has to be continued over a lengthy period, *e.g.*, in puerperal septicæmia, this mode of giving it is worthy of trial.

But under some circumstances a stimulation to the production of special mediators to anticipate disease may be indicated. For smallpox it is indeed already in general use, in the form of vaccinia-inoculation. It would be a good thing to do the same with tuberculin in individuals exhibiting a predisposition to tuberculosis; and as time goes on, it will no doubt be possible to extend such prophylactic treatment to the few other diseases which, like smallpox in the past and tubercle in the present, are the chief abbreviators of human life.[1]

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[1] Some remarks on the practical application of sera will be found in THE PRACTITIONER for October, 1902. This article obviously makes no pretence to completeness from either a practical or theoretical point of view.

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DISEASES OF CHILDREN: A REVIEW OF RECENT WORK.

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INFANT-FEEDING.

In any general survey of progress in the department of diseases of children some reference to infant-feeding must almost necessarily find a place, for of all the causes of disease in infancy and childhood there is none which is responsible directly or indirectly for such a vast amount of infantile suffering or for so appalling a mortality as the faulty feeding of infants. It has been shown by statistics (*Lancet*, Aug. 1902) that, whilst the annual death-rate amongst the general population is decreasing, the death-rate amongst infants was actually heavier in the ten years ending in 1900 than it was in the previous ten years; and there is reason to believe that this increase of infantile mortality is due to a wider prevalence of artificial methods of infant-feeding.

If it be true that in this country women are not only less willing, but actually less able, to suckle their infants than they were a generation or two ago, such a state of things calls for serious consideration by all who are interested in maintaining the physical standard of the nation. In a recent editorial in the *British Medical Journal* (May 30, 1903) the following statistics taken by Dr. M. Richards at Chesterfield were quoted, showing the disastrous effects of artificial feeding by a comparison of the diarrhœa-mortality amongst infants fed artificially with that amongst infants partially or entirely breast-fed.

Mode of Feeding.	Deaths from Diarrhœa, per cent.
Breast only	11
Breast and foods	22
Cow's milk and foods	67

"The importance of artificial feeding in increasing infantile mortality," the writer says, "cannot easily be exaggerated," and he agrees with the general opinion that "in connection with the more general urbanisation of the English population, the increased employment of young mothers in occupations away from home, and the unfortunate reluctance of many mothers to discharge their primary maternal duties, there has been a reduction of the proportion of children who receive the immense advantage of breast-food." Dr. C. G. Kerley, of New York, in a discussion on infant-feeding (*Jour. Amer. Med. Assoc.*, June 20, 1903) threw doubt upon the unwillingness of mothers to suckle their infants in that city; he thought that to insist upon continuous suckling was to put too severe a strain upon the maternal instinct; and he was in favour of giving the mother more freedom by allowing one artificial feed each day, so that the young mother may not be entirely debarred from recreation, whether it be at the opera, the matinée, or elsewhere. Such liberty, he considers, makes the nursing more successful, and enables the mother to nurse her child for a longer period; and, moreover, is of service when the mother is obliged by any emergency to leave her child.

The wisdom of introducing even the (perhaps small) degree of risk which attaches to one artificial feed daily might be questioned, but a greater objection to this as a routine practice, is the probability that it will lead to further laxity, and that the mother will take the supposed harmlessness of one artificial feed as a justification for further omission of breast-feeds. Undoubtedly the greater the proportion of artificial feeds daily, the greater the risk to the infant, and it is to be feared that the statement made by H. M. McClanahan (*ibid.*) in the same discussion, holds good for this country also—that too often a mother who has too little milk for her child is told by the doctor to wean the infant, whereas by supplementing the breast-feeding with a few artificial feeds daily, the suckling might be continued, and the risks of artificial feeding to that extent diminished. But it is less amongst the well-to-do than amongst the poorer classes that infants suffer from lack of the mother's milk, and the reason of this lies partly in the struggle for existence which necessitates the mother's going out to work to earn her own livelihood, and so makes suckling, at any rate by day, an impossibility. To obviate this difficulty Dubrisay (*Revue de Thérapeutique,* Aug. 1, 1903) suggests that municipal authorities should make regulations enabling women to keep their infants near them when at work, but he offers no suggestion as to how this is to be accomplished.

Febrile illness on the part of the mother during the few weeks after delivery, especially where the pyrexia is due to any infective condition, is often considered a reason for immediate weaning; but M. Perret (*Le Progrès Médical,* May 23, 1903) shows by a series of observations that even a temperature of 104°—105° F. in the mother is not necessarily an indication for the interruption of breast-feeding. Puerperal infection, broncho-pneumonia, tonsillitis, influenza and lymphangitis were amongst the maternal conditions in which suckling was continued, sometimes with a slight loss of weight in the infant at first, but usually with a steady gain of the child's weight during the greater part of the maternal illness. Precautions were taken to prevent infection of the infant; the mother's breast was washed with a weak solution of corrosive sublimate and then with boiled water before each suckling, and especially in cases of influenza the infant was kept away from the mother's room in the intervals between suckling.

Where breast-feeding is impossible, it is generally agreed that cow's milk, or the milk of some other animal, is the proper substitute; but as to the modification which is necessary there is still considerable divergence of opinion. There are those who hold that no modification whatever, unless it be the addition of a little sugar, is required, and that an infant three weeks old may have cow's milk just as it comes from the udder. Hauser, in an excellent summary of recent work on infant-feeding (*Fortschritte der Medicin,* Aug. 15, 1903), remarks somewhat cynically that the results which have been recorded from this particular method of feeding only prove how long-suffering the healthy stomach of a child may be, and quotes Biedert (*Arch. für Kinderheilk.* xxxii.) as his authority for the statement that defective nutrition and consequent liability to infective processes result from such concentrated feeding.

At the present time, however, the tendency is certainly not to any neglect of modification, but rather to the multiplication of minutiæ, many of which are at least of doubtful expediency. The modification of milk by dilution with water or whey, with addition of sugar, so as to obtain any desired percentages of proteïd, fat and sugar, is simple enough; the difficulty is to obtain not the percentage which the doctor desires, but the percentage which will meet the needs of the infant, and this bears no constant relation to age or weight, but varies with the idiosyncrasy of the particular child. T. S. Westcott (*Archives of Pediatrics*, Nov. 1902) discusses the management of the *fat-percentage,* which may easily give trouble if it is too high; he mentions a case in which not more than about 2 per cent. of fat could be digested, and considers that in many cases where there is chronic catarrh of the stomach or of the upper part of the intestine, unusually low percentages of fat are required. For this purpose he recommends simple dilution of milk with fat-containing whey (*i.e.* whey made from whole milk, not from skimmed milk) without any addition of cream.

The *proteïd-percentage* is probably more often at fault than the fat; and whilst by far the commoner fault is an excess of proteïd, especially of caseïn, the possibility of a serious deficiency is emphasised by V. Adriance (*Archives of Pediatrics*, Aug. 1903). As a result of the common knowledge that gastro-intestinal disturbance in infancy is often due to excess of caseïn, both mothers and doctors are apt to dilute milk to such an extent that the infant is actually starved of proteïd food. The symptoms of proteïd starvation are those of backward development, delayed dentition, late closure of sutures and fontanelles, fretfulness, and a tendency to perspire about the head—in fact, the symptoms of rickets. Another result of deficiency of proteïd is diarrhœa with anæmia and lowered vitality. Chapin also (*Archives of Pediatrics*, July, 1903) lays stress on the harm which may be done by giving too low a proportion of milk-proteïds, and suggests that where a sufficient quantity of these substances cannot be digested, they should be supplemented by other forms of nucleo-albumin. Elsewhere he has stated that 1·5 per cent. is the lowest proportion of proteïd which can safely be given to an infant four or five months old, whilst Adriance (*loc. cit.*) considers that at four months an infant requires 2 per cent. of proteïd.

Buttermilk is still being advocated as a food for infants, especially where there is chronic gastro-enteritis. Its composition would seem to defy all the modern canons of infant-feeding; nevertheless in practice it has been found to be of considerable value. H. Schwartz at the New York Academy of Medicine described a preparation of buttermilk which was made from centrifugal cream infected with lactic-acid bacilli and then kept in a warm room for eighteen hours before churning. To one litre of this buttermilk about 20 grms. of prepared flour are added, and about 40 grms. of cane-sugar; the mixture is boiled for five minutes and then kept on ice. The resulting food is of course highly acid, and contains albumen, 3·4 per cent.; fat, ·5 per cent.; carbohydrates, 10·3 per cent. It causes slight fever, which, however, passes off in a day or two without doing any harm; and infants thrive upon it. Such buttermilk is specially of value in fermentative and fatty diarrhœa, and in various intestinal disorders after the acute stage has passed.

G. Jacobson (*Arch. de Méd. des Enf.*, Feb. 1903) considers that buttermilk is useful even for healthy infants, where breast-feeding is impossible, but especially in cases of gastro-enteritis and indigestion. Its value lies chiefly in its easy digestibility which is due chiefly to its acidity, the low percentage of fat, and the extremely fine division of the already coagulated caseïn; it has, moreover, the advantage of being inexpensive.

It is evident that the supposed necessity for alkaline reäction in food for infants is disproved by the success of such a food as buttermilk; but according to Kerley, Gieschen, and Myers (*Med. Record*, Aug. 8 1903), the very evidence on which the theory of alkalinity is usually based has now been disproved. By a series of observations with phenol-phthalein, which is a much more delicate indicator than litmus, these observers found that human milk is normally acid, not alkaline as has been taught; and therefore the alkalinising of cow's milk, which is also normally acid, is not necessary, if it is merely desired to imitate the reäction of human milk. The addition of lime-water or bicarbonate of soda to cow's milk retards or inhibits the formation of curds by rennet, and it is on this ground, not because they are antacids, that their addition may be useful.

There is considerable difference of opinion as to the practical value of the milk of animals other than the cow in infant-feeding. Whilst the use of *goat's milk* has been strongly advocated, for instance by Barbellion (*Bull. de l'Acad. de Méd.* March 1902), on the ground of the rarity of tuberculosis in goats, and the easy digestibility of their milk, cases of choleraic diarrhœa have been reported from the use of this milk unboiled (Zammit, quoted by Hauser, *Fortschritte der Medicin*, Aug. 1903). Nobécourt and Merklen (*La Presse Médicale*, Dec. 1902) showed also that goat's-milk lacks the amylase and the salol-splitting ferments which are present in human milk. In this respect asses' milk approximates most nearly to mother's milk; and Ranke (quoted by Hauser, *loc. cit.*) points out that the *relative* proportions of the various constituents of asses' milk correspond closely with those in human milk, and to this he attributes the success which attends its use during the early months of infancy. In the later months of infancy it is less successful, for its fat-percentage is too low.

At what age an infant should begin to take cereal or other food in addition to milk is not always easy to determine. A. D. Blackader (*Montreal Med. Jour.*, Vol. xxxi. No. 7) states that an infant is born with a high percentage of hæmoglobin, and that owing to the low proportion of iron in milk, the hæmoglobin gradually diminishes so long as the infant is fed on milk alone. He would add cereals, and meat-juice or broth, to the diet at the tenth or the twelfth month. Oatmeal properly cooked is very useful, as it is one of the richest in iron of the cereals, and after the age of twelve months the yolk of an egg lightly boiled is a valuable food on account of its richness in fat, lime-salts, and the organic compounds of phosphorus and iron. W. M. Hartshorn (*Med. Record*, June 27, 1903) would give cereals at the age of seven months, and after trial of flaked rice, cream of wheat, oatmeal gruel, and farina, came to the conclusion that none of these were so satisfactory as bread-and-milk. This was prepared by boiling stale bread until it was thoroughly softened, then pouring off the water, adding milk, and boiling for three or four minutes; the quantity of this food was increased gradually from one teaspoonful a day up to two or three ounces daily. With this addition to the simple milk-feeding, the infants were found to gain weight more rapidly.

SUMMER DIARRHŒA.

In connection with the summer diarrhœa of infants, perhaps the most interesting of recent observations are those on the presence of the Bacillus of Dysentery (Shiga's bacillus) in these cases. S. Flexner, in an interesting paper read before the American Pediatric Society (*Pediatrics*, July, 1903) stated that this bacillus was found in 45 out of 52 cases of severe summer diarrhœa; the blood-serum of these children gave a positive reäction with the organisms obtained from their own dejecta, and also with organisms obtained from cases of dysentery in Japan and the Philippines. The bacillus is found most easily when mucus, or blood and mucus, are present in the fæces. L. E. Holt (*loc. cit.*) had had 120 cases of diarrhœa examined bacteriologically, and only 37 shewed the Shiga bacillus and gave the blood-reäction. Every case with blood in the stools yielded the bacillus, but many in which there was much mucus without blood did not show the bacillus. The bacilli may be found in the stools as long as six weeks after the acute symptoms have subsided, but usually they disappear much sooner, and the blood-reäction is absent after the second week. There is no characteristic lesion in these cases. Flexner prepared a serum which was protective to guinea-pigs, and Holt used this for infants with some good results; the effect, however, was very transient, so that the dose had to be repeated frequently.

CONGENITAL HYPERTROPHY OF THE PYLORUS.

Congenital hypertrophy of the pylorus has come into prominence during the past few years, chiefly, no doubt, because what appeared at first to be a hopeless condition has now been shewn to be amenable both to medical and to surgical treatment. Cautley and Dent (*Lancet*, December 20, 1902) recorded two cases successfully treated by pyloroplasty, and collected eight other cases in which operation had been successful. In the discussion on their paper at the Royal Medico-Chirurgical Society the opinion was expressed that in some cases recovery occurred without operation, and cases were cited to prove this. H. W. Gardner (*Lancet*, January 10, 1903) has since published another case of recovery with careful dieting, in an infant who showed the characteristic peristalsis and pyloric tumour. It is clear, however, that there is some difference of opinion as to the symptoms which justify a diagnosis of congenital hypertrophy of the pylorus. G. Variot (*Gaz. des Hôpitaux*, June 16, 1903) relates a case which he says the English would include under this heading; it was a case of troublesome vomiting in an infant, with slight dilatation of the stomach, as determined by percussion; no other signs were present. In two cases recorded at the New York Academy of Medicine (*Archives of Pediatrics*, August 1903) infants with symptoms resembling those of chronic gastric catarrh were treated by operation and died shortly afterwards; the stomach and pylorus in each case showed some thickening. Cases have been recorded in which children past the age of infancy have shown stenosis of the pylorus, apparently the result of hypertrophy. Whether these should be classed with the infantile cases of so-called congenital hypertrophy of the pylorus is perhaps doubtful. F. W. Shaw (*Brooklyn Med. Journ.*, May 1903) records the case of a boy, aged 5 years, who had suffered since he was an infant with recurrent attacks of abdominal pain and vomiting, lasting each time for several days: finally he succumbed to an attack which lasted 10 days; at the autopsy the pylorus was found to be hypertrophied.

RECURRENT VOMITING.

Recurrent attacks of vomiting in children past the age of infancy are rarely due to any such mechanical cause; and since Gee first described recurrent vomiting as a disorder "sui generis" in children, there has been great uncertainty as to its pathology. Marfan (quoted in *Boston Med. and Surg. Journ.*, June 25, 1903) in 1901 recorded the association of acetonæmia with recurring attacks of vomiting: the child's breath and also the fresh urine smell strongly of acetone, and the characteristic acetone reäction is obtained in the urine. Recently several American writers have confirmed this observation. D. L. Edsall, at the Philadelphia Pediatric Society, reported six cases in which this evidence of an acid intoxication was observed; and E. L. Pierson (*Archives of Pediatrics*, July 1903) records three cases in which acetone and diacetic acid were found in the urine of children during the attack. The symptoms in all these cases are much alike; vomiting begins without obvious cause and lasts for two or three days, and sometimes longer; one case vomited 84 times in 44 hours, but usually the vomiting is less severe than this. The temperature in all Pierson's cases was raised, in one to 103·4° F. The attacks varied in frequency; in two of these cases they recurred about once in three months. H. L. K. Shaw (*Archives of Pediatrics*, November 1902) states that constipation generally immediately precedes or accompanies the attacks, and mentions the finding of albumen and casts in the urine during the attacks in some cases. Some tenderness of the abdomen at this time has also been noted, for instance in a case recorded by M. L. Guinon (*Gaz. des Malad. Enf.*, January 1903).

The prognosis is almost invariably good. Shaw (*loc. cit.*) states that there are only five fatal cases on record. Treatment has hitherto been most unsatisfactory; the rectal injection of large quantities of normal salt-solution, recommended by Marfan, has no effect on the vomiting, although it may allay the resulting thirst; nothing has hitherto been found effectual, and in most cases the attacks run their course, uninfluenced by feeding or by drugs. A practical interest, therefore, attaches to the suggestion made by Edsall (*Amer. Journ. Med. Sciences*, April 1903) that large doses of diffusible alkali should be administered at short intervals. For this purpose he thinks sodium bicarbonate, given hourly in moderate doses until 2 drachms have been used, is more effectual than "milk of magnesia." If the sodium bicarbonate is given directly the child shows any of the prodromal symptoms (lassitude, constipation, headache, loss of appetite) which are present in some cases, the attack may be aborted altogether, and even if the vomiting has already begun early administration of sodium bicarbonate will sometimes arrest it.

FUNCTIONS OF THE THYMUS GLAND.

The part played by the thymus gland in diseases of children has given rise to much discussion. On the one hand it has been held that its functions, whatever they may be, are evidently of little or no practical importance; on the other hand, cases are frequently recorded in which death is attributed to abnormal conditions of the thymus. Quite recently Stokes, Ruhräh, and Rohrer (*Amer. Journ. Med. Sci.*, November, 1902) have suggested that there is some causal relation between atrophy of the thymus and infantile marasmus. They found that from a microscopic examination of this gland they could estimate the degree of marasmus in the infant; and in sixteen out of eighteen cases of fatal marasmus they found that the weight of the thymus was far below the normal (the average in sixteen cases was only 2·2 grammes, whereas the "general normal weight" is 18 grammes). They gave thymus gland in doses of 1–3 grains three times a day to marasmic infants, but without appreciable benefit. The thymus gland has been credited also with a share in the production of rickets, and Mendel (*Münch. Med. Woch.*, January, 1902) accordingly dosed with thymus a hundred children who suffered from rickets, and attributes improvement to the treatment. Sudden death in children has frequently been associated with an unusually large thymus, but the relation, if there be any, between the size of the gland and the fatal event remains uncertain. E. C. Jones (*Archives of Pediatrics*, August, 1903) records the case of an infant, aged 3½ months, who, after dyspnœa with some cyanosis, and distress, was found dead about nine hours after the onset of symptoms. The thymus gland was found to be much enlarged, but so also were the lymphatic glands in the mediastinum, mesentery, and inguinal regions, and both liver and spleen were enlarged. It is suggested that this is one of the cases of "status lymphaticus," a condition in which, with general enlargement of lymphatic glands, spleen, and thymus, sudden death occurs, not from any mechanical cause, but from a profound disturbance of nutrition. At the same time the possibility that here, as in some other recorded cases, the large thymus may have caused death by direct pressure on the great vessels and heart can hardly be disproved.

Wilson (*Archives of Pediatrics*, September, 1903), discussing disturbances of respiration in the newborn, states that attacks of dyspnœa in the newborn may be associated with hypertrophy of the thymus; these attacks he considers to be convulsive, not the result of direct pressure.

As an instance in which direct pressure by an enlarged thymus seemed to be the cause of sudden death in an infant may be mentioned a case recorded by A. Caillé (*Archives of Pediatrics*, March, 1903). The infant was six months old; there was a loud systolic murmur at the base of the heart; convulsive seizures had occurred twice; the infant died suddenly, and a large thymus was found which was thought to have compressed the great vessels. Jacobi, discussing this case, pointed out that in one case recorded by Koenig suffocative symptoms in an infant had been relieved after the thymus had been drawn out and fixed in such a way that it could not press upon the blood-vessels or air-passages, and as an aid to diagnosis he stated that the dulness due to an enlarged thymus was most easily appreciated by percussion from below whilst the child was supported face downwards.

SCURVY.

Infantile scurvy has been so fully described by Cheadle and Barlow that it is hardly likely that much can be added to our knowledge of its clinical symptoms and morbid anatomy. Its rarity in Switzerland might be argued from an article by Hagenbach - Burckhardt (*Corresp.-Blatt. f. Schweiz. Aerzte*, December 15, 1902).

This writer finds only seven cases recorded in Switzerland, and adds to these the first case which has been observed at the Children's Hospital in Basle. He mentions also the extreme rarity of infantile scurvy in Russia, while adult scurvy is by no means uncommon there. It may be suspected, however, that the apparent rarity of scurvy in some places depends partly on failure to recognise the disease. J. Comby (*Revue de Thérapeut.*, May 1, 1903) relates the case of an infant, aged 19 months, with typical infantile scurvy, who was first treated as a case of acute rheumatism, and then fixed in an apparatus for supposed hip-disease, and subsequently considered as suffering with pneumococcal arthritis, or possibly with infantile paralysis. T. M. Rotch (*Med. News*, September 12, 1903) records a case in which the unfortunate infant not only had incisions made into both legs, but also had the entire shaft of one tibia removed, under the impression that the case was one of acute osteomyelitis; whilst in another case an amputation for supposed osteo-sarcoma had been suggested.

The pathology of infantile scurvy still remains obscure, and it is evident that the misleading name which is sometimes applied to it, "scurvy rickets," tends to involve it in still greater obscurity. Ausset (quoted in *Arch. Pediatr.*, May, 1903) discusses the relation of this disease to rickets, and comes to the conclusion that it is a hæmorrhagic variety of rickets, and that it is due to a similar ætiological factor; the absence of any evidence of rickets in the bones he considers as of no importance, as the bony deformities are to be referred to gastro-intestinal disturbances of more chronic duration.

It has been suggested by Dr. W. B. Ransom (*Brit. Med. Jour.*, February 22, 1902) that infantile scurvy is due to products of decomposition in the food, and that the fault lies not in the sterilisation or drying processes in the preparation of infant foods, but in the presence of ptomaines or other toxic substances from contamination during prolonged keeping or from insufficient sterilisation of the food.

TUBERCULAR PERITONITIS.

The treatment of tubercular peritonitis has been much discussed within the past few years, and whilst there is a consensus of opinion that there are cases in which surgical measures may be of value, there would seem to be a growing feeling that such cases are comparatively uncommon, and that the recoveries which have been ascribed to laparotomy are probably in many cases to be attributed rather to improved diet, general hygiene, and change of air which usually form part of the treatment, than to any special virtue in the operation. L. Guthrie (*Arch. Pediatr.*, April 1903) says that in his opinion "simple laparotomy is useful only in so far as it lets out fluid. Dry cases, which are also supposed to be cured by the operation, would, I believe, recover equally well if let alone, provided that no complications such as extensive ulceration, septic peritonitis due to suppuration of glands or perforation, meningitis, or advanced pulmonary phthisis occurred. Indeed the recovery of uncomplicated dry cases, not operated upon, compares favourably with laparotomy cases where convalescence is retarded by abscesses and fistulæ arising from tuberculous infection of the operation scar." Guthrie points out also that even when there is fluid, it tends to subside spontaneously, and that in acute cases surgical interference is unadvisable unless the fluid is causing great distension and distress; for, if removed, the fluid will speedily reäccumulate. It is only in chronic cases, where the fluid fails to disappear spontaneously, that any surgical interference may be needed, and then a small incision, large enough to admit a blunt trochar, may be quite sufficient; a more extensive laparotomy is only to be done where these measures fail to prevent reäccumulation of fluid.

There can be little doubt that the exaggerated value which has been put upon laparotomy as a treatment for uncomplicated tubercular peritonitis is due largely to the fact that the tendency of this disease to spontaneous recovery under favourable hygienic conditions has been ignored. G. A. Sutherland (*Arch. Pediatrics*, February 1903) says that "perhaps in no form of tuberculosis does nature make greater or more successful efforts to cope with the disease." Out of 27 cases treated medically, 22 (81·3 per cent.) recovered, one was unrelieved, 4 died; out of 14 cases treated surgically, 7 (50 per cent.) recovered, and 7 died. Nineteen of the cases recorded as "recoveries" had been kept under observation more than one year. His conclusions as to progress are as follows:—(1) In uncomplicated tubercular peritonitis the prognosis is good. (2) When tubercular pleurisy is present, the prognosis is still favourable. (3) The prognosis is rendered less favourable in the case of a strong family history of tuberculosis, an infancy passed under bad hygienic and dietetic conditions, a constitution of feeble resistant power, or a history of severe infective illness in early life. (4) The prognosis is rendered less favourable in the presence of one or more of the following symptoms:—Continuous pyrexia, rapid wasting, persistent diarrhœa, rapid pulse, or recurrent acute exacerbations. (5) The prognosis is rendered less favourable in the presence of one or more of the following local complications: (*a*) tubercular ulceration of the bowel, (*b*) extensive caseation of the mesenteric lymph-nodes, or of tubercular masses, (*c*) localised suppuration from infection through lymph-nodes or the intestine, (*d*) obstructive symptoms from bands or matting of the intestine. (6) The prognosis is bad in the case of the following complications: (*a*) The rupture of a suppurating lymph-node, or the perforation of an intestinal ulcer into the peritoneal cavity, (*b*) pulmonary tuberculosis, (*c*) tubercular meningitis, (*d*) general miliary tuberculosis. (7) In tubercular peritonitis the prognosis is not appreciably affected by simple laparotomy.

Amongst the cases mentioned by Sutherland is one in which the abdomen, which had felt "like plaster of Paris" owing to fibro-ascitic tubercular peritonitis, became quite normal after the child had been sent away to the country. C. Leroux (*Annales de Médecine et Chirurg. Infantiles*, September 15, 1903) describes a very similar case in a girl, aged 11 years, whose abdomen was hard and distended, with masses of tubercular material to be felt, and enlarged veins to be seen on the abdominal wall. With a prolonged stay at the seaside, and the administration of guiacol, the child recovered completely and the abdomen became perfectly supple and normal. Leroux emphasises the value of prolonged residence at the seaside for such localised forms of tuberculosis, and says that to obtain the best results the seaside treatment must be begun early and continued without interruption until recovery is complete.

CONGENITAL SYPHILIS.

Congenital syphilis is hardly less protean in its manifestations than the acquired disease. According to Abt (*Journ. Amer. Med. Assoc.*, January 31, 1903) many of the cases of spontaneous hæmorrhage from various parts of the body in the newborn are the result of congenital syphilis, and he quotes observations which demonstrate characteristic changes such as endarteritis in the blood-vessels, especially in the medium-sized vessels in the submucosa of the alimentary tract. In a case of melæna neonatorum with congenital syphilis Esser (*Archiv f. Kinderheilk.*, quoted in *Pediatrics*, November 1, 1902) found in addition to osteochondritis and arteritis, a syphilitic enteritis, the small intestine showing great thickening and ulceration of its mucosa.

The relation of congenital syphilis to *nephritis* in children has been emphasised by several writers recently. An acute interstitial nephritis was found in 2 infants, aged 16 months and 8 months respectively, by Sutherland and Walker (*Brit. Med. Journ.*, April 25, 1903), and one can hardly doubt that such a condition must be the initial stage of a chronic granular kidney such as is occasionally found in childhood and even in infancy.

But the evidence that chronic interstitial nephritis in early life is commonly due to syphilis is by no means conclusive.

In 24 cases of chronic interstitial nephritis in children and young adults, J. E. H. Sawyer (*Birmingham Med. Rev.*, August 1903) found good evidence of congenital syphilis only in one, and a suspicion of syphilis in two others; but, as he mentions, an undoubted case of syphilitic chronic interstitial nephritis in an infant has been recorded by Massalongo. It is noteworthy that Sawyer's only case of proved syphilis was in a girl of 18, not in a child, although it is possible that here also the nephritis may have begun in childhood.

The significance of *enlargement of the spleen* as evidence ot congenital syphilis is discussed by A. B. Marfan (*Annales de Médecine et Chirurg. Infant.*, May 15, 1903). According to Gee, about a quarter of the cases of congenital syphilis show enlargement of the spleen, whilst other observers have placed the frequency as low as 10 per cent. and as high as 78 per cent.; Marfan himself finds splenic enlargement in half the cases of congenital syphilis. He considers that syphilis is by far the commonest cause of enlargement of the spleen in infancy, and that when the spleen is affected in this way there is almost always a greater or less degree of anæmia and often also some increase in the size of the liver, and of lymphatic glands; such a combination may be the only evidence of syphilis.

Undoubtedly rickets alone may also cause enlargement of the spleen; but even if rickets is present the splenic enlargement should always suggest a careful inquiry for syphilis, for the two diseases are not uncommonly combined, as his own statistics showed; amongst 40 cases of splenic hypertrophy there were 12 in which the association of these two diseases seemed probable, and 7 in which the association was certain.

The so-called "pseudo-paralysis" described by Parrot as a symptom of congenital syphilis is the subject of a paper in the *Jahrbuch für Kinderheilkunde* by F. Scherer (*Annales de Méd. et Chirurg. Infant.*, August 1, 1903). In 50 cases of hereditary syphilis he found pseudo-paralysis 11 times, *i.e.*, in 22 per cent. In 6 out of the 11 cases this symptom appeared during the first 3 months of life. As a rule it takes the form of a brachial monoplegia; in some cases there is an obvious cause in a lesion of the bone, but in others the cause is obscure, and Scherer suggests that a syphilitic toxine, presumably by its effect on the nerves, may be the cause.

Malnutrition is a well-recognised result of congenital syphilis. According to C. G. Kerley (*Med. Record*, December, 1901) not only infantile marasmus, but also a persistent malnutrition in children between the age of three years and puberty, may be traceable to this cause. In some cases, as he points out, rapid improvement follows the administration of mercury and iodide; but it is to be remembered that absence of improvement under such treatment is no evidence of absence of syphilis, for, as Coutts pointed out in his Hunterian lectures, many cases of marasmus in early infancy, as the result of undoubted congenital syphilis, waste persistently in spite of vigorous anti-syphilitic treatment. It is of great importance that such cases should be kept at the breast. J. E. Winters (*ibid.*) stated that in a large experience he had never known an infant with hereditary syphilis to infect its mother. A. Jacobi, however (*ibid.*), stated he had himself seen exceptions to this rule, and others had been reported. If this occurrence can be proved, it must certainly be of extreme rarity, and Jacobi was of opinion that a baby with congenital syphilis should be suckled by its mother, who should be warned of the possible though not probable danger. The extremely minute risk to the mother is outweighed by the great gain to the infant, especially where syphilitic marasmus is present.

PYELITIS.

Amongst the urinary disorders of childhood pyelitis has recently attracted attention as a cause of acute illness in infancy, with symptoms which may be extremely misleading unless the occurrence of this condition is recognised. Dr. John Thomson (*Scottish Med. and Surg. Journ.*, July, 1902) has given an excellent description of acute pyelitis in infancy, and lays stress upon the occurrence therewith of rigors, which are extremely rare in any other disease of infants. The temperature reaches a high level, often above 104° F., and is usually more or less remittent, so that the chart may resemble that of typhoid. There is usually marked restlessness and distress, and sometimes apparently colicky pain. The urine is acid, with a small amount of albumen and a considerable quantity of pus, sometimes enough to make the urine turbid. Of eight cases recorded by Dr. Thomson all were girls, and another case in a female infant is reported by Dr. J. Ritchie (*ibid.*). But such a condition is certainly not limited to the period of infancy or to girls. Dr. W. C. Bosanquet (*Edin. Med. Journ.*, August, 1903) has drawn attention to the occurrence of attacks of pyrexia in older children as a result of pyelitis, and reports two cases, one in a boy aged 2½ years, the other in a girl aged 6 years. In the former there were rigors, and in both there was sickness with the pyrexia, and the child seemed ill. The bouts of pyrexia recurred in the younger child every four or five days; in the girl they recurred every ten or fifteen days, and in the intervals she appeared quite well. The urine in each case showed a small amount of pus. Dr. Bosanquet emphasises the necessity for careful examination of the urine in obscure cases of pyrexia in children, especially where the pyrexia tends to recur. In his cases neither the alkaline remedies, which Thomson and others have recommended, nor urotropine seemed to have any decided beneficial effect. One of his cases appeared to recover finally after, or in the course of, scarlet fever.

A REVIEW OF RECENT LITERATURE IN THE FIELD OF DENTAL SURGERY.

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DENTAL ANÆSTHETICS.

THE third and fourth volumes of the proceedings of the Third International Dental Congress, published this year, contain some interesting dental reading.

Dr. Dudley Buxton, in a Review of Anæsthesia, maintains that gas is still the best of dental anæsthetics for shorter operations, and ether for longer. Chloroform he condemns: deaths from chloroform are met with among the most healthy patients, due to vasomotor paralysis and the collection of the blood in the venous sinuses ("abdominal pool" of Hill), leaving the nerve-centres bloodless and the heart unable to maintain a circulation. The same applies to bromæthyl and pental.

While on the subject of anæsthetics, it may be mentioned that opinions are being expressed—as witness some letters lately appearing in the *British Medical Journal*—that somnoform is not so safe an anæsthetic as it is said to be. Its danger is by some rated as about that of bromide of æthyl (about one death to 4,500 cases), and, as insisted on by Dr. Hopson (*British Dental Journal*, January, 1903), it is often followed by disagreeable feelings of sickness, giddiness, &c. In any case it is emphatically not an anæsthetic to be given by a dentist alone.

As to the method of action of somnoform, Dr. George Holland (*British Dental Journal*, October, 1903) finds by experiment that it acts on the nerve-cells of the cerebellum—first on Purkinje's cells, and later, after saturation by the anæsthetic, on the pyramidal cells. Figures are given showing the very definite microscopic effects observed.

Dr. Hewitt, at the annual meeting of the British Dental Association, 1903, proposed a new method of anæsthesia, to obtain insensibility of about one minute's duration, by the use of gas followed by ethyl-chloride. Not much gas is used, and the ethyl-chloride is injected into the gas-bag, after a certain amount of gas has been administered (partly by rebreathing) from a glass tube fixed into the inlet-tube at the bottom of the bag. He recommends it as probably a useful method in the hands of experienced anæsthetists.

DENTAL PATHOLOGY.

Returning to the proceedings of the Dental Congress, Dr. A. Gysi suggests that the *sensitiveness of dentine* is due, not to actual nerves in the dentinal canaliculi, but to transmission of pressure through their protoplasmic contents, which, being rich in water, act practically as columns of this very incompressible fluid and transmit pressure direct to the highly nervous pulp.

Dr. S. Bernheim thinks that *tuberculosis of the gums* and alveolar border is of more frequent occurrence than is generally admitted, and quotes figures in support of this view. A dento-alveolar origin he thinks possible in some cases; in others the disease is systemic, and finds a secondary focus in the mouth.

Concerning the *stomatitis of albuminurics*, Chas. Lee thinks that it begins round carious teeth or roots, and could always be prevented by proper attention to cleanliness of the mouth; the chronic disease acts only as a general predisposing cause.

Pyorrhœa alveolaris has recently been much discussed. Divergent views and, as a consequence, methods of treatment are advocated. Some regard it as entirely constitutional, and one dental surgeon records two cases cured by five weeks at Carlsbad after failure by local methods. Others are convinced of the truth of the local view, and methods of treatment according to this view are constantly being put forward. All are more or less variants on the anti-bacterial theory, and of varying thoroughness or heroism. Minutely careful removal of tartar and disinfection, and scraping or cauterising of the peri-dental pockets are the keynotes. Fixation of loose teeth by bridge-work is well spoken of, but the bridge-work must extend to several teeth and be well free of the gum.

C. S. Tomes, in an editorial article in the *British Dental Journal*, proposes a careful study of the flora of the mouths of animals—*e.g.*, dogs, which are almost immune to caries—as a means of obtaining a clue to the conditions needed to establish immunity in the human mouth. And, in relation to pyorrhœa alveolaris, he notes that in the same house in the London Zoological Gardens, lemurs and monkeys are kept side by side and fed on almost identical food, and yet the lemurs suffer extensively from pyorrhœa and the monkeys almost never.

In more purely scientific or laboratory work, Dr. Miller, of Berlin, experimenting on *saliva in its relation to immunity* (from dental caries), concludes that human saliva has no appreciable bactericidal properties, nor do alexines from the blood make their appearance in it.

F. Neuman discusses *polypus of the tooth-pulp*, and especially a form not infrequently met with, which is covered with epithelium. This has been regarded by some as a true metamorphosis of connective-tissue elements into epiblastic forms, by others as a transplantation. He suggests that the transplantation is effected by the simultaneous growth into the cavity of a carious tooth of a pulp-polyp and of a gum-polyp, followed by blending of their apposed surfaces after scarification by the injuries of mastication, and this again by eventual separation by the same agency.

J. C. Oliver (*Annals of Surgery*, January, 1903) records three cases of *cysts in connection with teeth*, which are of considerable interest to pathologists.

Case 1, a man of 25, showed "considerable thickening and hardening of the maxilla," which was found to have its origin in a buried bicuspid, round which there was suppuration. Such a formation of new bone round a buried tooth is rare enough to allow of its possible origin being overlooked and a diagnosis of sarcoma or syphilis being made. (The patient was free from syphilis.)

Case 2, a child, of 10, exhibited a swelling round the unerupted permanent incisor and canine. Escape of fluid took place on incision, which revealed a reticulated interior. The crowns of the teeth projected into the "cyst." Microscopic examination showed the "typical picture of a myeloid sarcoma."

Case 3 was a child of 12; a swelling about the "size of an English walnut" was removed from the left lower jaw, together with some buried teeth (molars and bicuspids). The microscope showed it to be a giant-celled sarcoma. Two months later a cyst round the left permanent canine was operated on, and on microscopic examination the walls were found to be composed of "granulation tissue with inflammatory papillæ dipping into the interior of the cyst."

Case 2 looks as if it had been a myeloid sarcoma undergoing cystic degeneration, and both Case 2 and the first tumour in Case 3 may have had their origin in the irritation of broken-down temporary teeth, as suggested by the writer in the May number of this journal.

Commenting on these cases, and diverging into the region of dental cysts, Mr. Oliver says, "the almost universal opinion is that epithelium is not found in cysts connected with the roots of the teeth." Such an opinion may prevail in America, but here in England, and equally so on the Continent of Europe after the work of Maitôt, a cyst connected with the root of a tooth—a dental cyst—is believed to be a cyst of epithelial origin, and hence failure to find an epithelial lining is regarded as an accident and not as the normal occurrence. The writer has examined a large number of such cysts, and has never yet failed to find an epithelial lining.

ORAL CONDITIONS IN RELATION TO GENERAL HEALTH.

Considerable attention has been paid by general surgeons and physicians during the past year to mouth-conditions. The influence of *septic conditions of the mouth* in the production of local and general disease has been strongly insisted on and further elaborated. Mr. Roughton, in a series of papers in *The Lancet,* deals with the question, and relates in a later number the case of a nurse in whom the general pyrexia and other symptoms due to pyorrhœa alveolaris led to a mistaken diagnosis of typhoid fever—only revised when, further expected symptoms of typhoid failing to appear, the mouth was examined and treated. At first Widal's reaction was positive, but after a course of antiseptic mouth-treatment it became negative.

Dr. Harry Campbell, concluding a thoughtful series of papers on "Observations on Mastication" in *The Lancet,* in which he shows the need for more fibrous food both in growing and adult subjects, says of the tooth-brush. "The modern tooth-brush requires to be used with great caution, as it is capable of doing much harm, not only by removing the mucoid film, which according to Dr. Wallace, protects the teeth from corroding agencies; but probably also by injuring the edge of the gum and the neck of the teeth, and thus setting up the condition known as erosion. . . . The brush should be soft. . . . Tooth-powders should not be used as a matter of routine. . . . Antiseptic powders and washes are to be scrupulously avoided, for it is neither desirable nor possible to render the buccal cavity aseptic; myriads of bacteria flourish in it, many of which play a useful part as scavengers."

With *all* of this it is impossible to agree. Certainly tooth-powders should not be used as an everyday matter. Used with vigour they grind away the necks of the teeth, but this is not "erosion"; and the insoluble particles get lodged between the teeth and form foci for tartar. Nor is it always possible to render the mouth aseptic, but it *is* desirable. Many species of bacteria flourish, but it is not always the beneficent scavengers which have the upper hand: the survival or preëminence of one or other form is too much a matter of "soil," so easily altered by different foods, to render it safe to leave this experiment in sewage-farming to follow its course in the mouth.

Why, also, should not the tooth-brush be hard? We confess we not know of "corroding agencies" that are in such continuous action as to require a mucoid film left on the teeth to protect them; unless it be acid-forming bacteria, which will find a nidus in this very film.

Other writers take very different views from those of Dr. Campbell. In the *Philadelphia Medical Journal* (March 28, 1903, and April 4, 1903), Dr. D. D. Smith treats of septic mouth-conditions. Here in "the vestibule of human life," he urges, a greater sanitary reform can be made than all the local sanitary boards are capable of. In sneezing, coughing, licking letters, &c., the subject becomes a danger to others. He urges frequent oral toilet by competent operators—dentists—both as curative and prophylactic.

As a local result of such oral septicity trauma is accompanied by special dangers. J. A. Hofheimer (New York *Medical Record*) records a case of generalised pyæmia in a boy following fracture of the mandible in the attempt to extract a tooth. The boy recovered with the loss of half his mandible by necrosis; this was later largely reformed by the periosteum.

MM. J. and C. Tellier (*Lyon Médical,* February, 1903, pp. 189 and 235) have made a study of septicæmias of bucco-dental origin. They formulate a list of six varieties consequent on bucco-dental sepsis :—

1. Chronic septicæmia leading to "dental or buccal cachexia."
2. Acute generalised septicæmia.
3. Acute lymphatic septicæmia.
4. Venous septicæmia.
5. Septico-pyæmia.
6. True pyæmia with metastatic abscesses.

They call attention to the danger of setting up acute traumatic septicæmia by operating on a mouth already septic, and to some cases which they have observed of fatal syncope resulting from septicæmias of oral origin. Such syncope may occur without any warning in a patient whose progress is apparently entirely satisfactory, and is analogous to a toxic heart-failure in any other acute microbic disease. Whether a careful daily examination of the heart would lead to suspicion of such danger is a matter worth consideration.

To make the toilet of the mouth before operating it is not sufficient merely to have the mouth washed out with some antiseptic solution, the local cleansing of the teeth must be as carefully done as would that of the skin in a major operation; and in some cases it will be necessary to repeat this for several days before operating, both to relieve the local condition and to allow the patient to recover in some measure from the general toxic poisoning.

As a mouth-wash, Mr. J. Lederer (*Medical Record,* April 19, 1902) recommends a solution of chlorinated soda. The present writer always uses a lotion of carbolic acid and sodium bicarbonate in water (grains x. of each to the ounce of water) as a mouth-wash, and a stronger solution applied on cotton-wool pledgets thoroughly to clean the tooth to be extracted and its neighbours, especially round their necks. After extraction the socket should be syringed, and some writers recommend packing for a day; but in an ordinary case of extraction this seems inadvisable, since the socket rapidly fills up with an aseptic blood-clot which can be kept clean by frequent use of a mouth-wash.

Of considerable interest is a paper[1] by a French author, M. Jacquet, that *alopecia* is often of dento-reflex origin. He states that both as regards the side of the scalp attacked, and as regards time of onset, *alopecia* is frequently definitely related with the onset of dental troubles, and that in such cases local oral treatment produces good results on the alopecia. A possibility, however, of toxic origin is not insisted on.

The good results obtained in a case of *hæmophilia,* reported by Mr. F. Morley to the Odontological Society of Great Britain, are well worthy of note. By use of large doses of chloride of calcium internally for a week, and adrenalin locally on strips of gauze, kept applied for ten minutes, he was able to extract a tooth without loss of blood. The history of the case, and the fact that the gums had been bleeding for three days round the jagged and loose temporary molar, left no doubt as to the diagnosis.

MECHANICS OF THE JAWS.

Mr. Arbuthnot Lane, in a paper on "Some Points in the Mechanics of the Jaws," read before the Odontological Society of Great Britain, postulates three main agencies as concerned in the due development of the jaws :—

1. Passage of air through the naso-pharynx ;
2. Size and use of the tongue ;
3. Teeth, both by occupying space and by transmitting pressure.

He showed models of an infant born without any anterior nares, showing an abnormally high palatine arch, illustrating what may be laid down as a law, viz., that "the height of the palate varies inversely as the development of the naso-pharynx."

THE INCREASE OF DENTAL DISEASE.

The Lancet (Nov. 15, 1902), in an editorial under this heading, calls attention to the seriousness of this question. In this country (England) it is computed that about 86 per cent. of children have defective dentitions, while on the Continent the percentages in some cases average 96 and even 98·75. The deciduous teeth are equally prone to decay with the permanent, and serious injury to nutrition results during the whole growing period. Experience of early care of the teeth—filling temporary teeth where possible—points to the fact that such children suffer less from " adenoids and other ailments "—a fact of supreme importance in every aspect. Dr. Kingston Barton quoted in the same article, after experience of 20 years, is of opinion that caries is far less prevalent in children brought up at the breast, and in a paper read at the Third International Dental Congress (*Transactions*, Vol. III.), T. Frick upholds the same opinion. He includes sterilised cow's-milk in the list of artificial substitutes for human milk. Actual experiments on dogs, carried out by an American observer, show that artificially-fed pups develop badly-formed teeth, like the rachitic teeth of man.

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[1] Quoted in the *Journal de Médicine et Chirurgie Pratiques*, February 10, 1903, Art. 19,715.

Dr. Frick states that children who are breast-fed for three or six months possess better teeth than those who have never taken the breast, and concludes that the longer the breast is taken the better will be the teeth. He thinks that, in general, all the tissues of the body suffer from artificial feeding, but that the teeth especially deteriorate, showing a modification of form which allows of more ready lodgment of débris and a diminution of salts of lime.

We may suggest that the "special" incidence of the results of bad feeding on the teeth is only apparent. All the tissues suffer, but of them all the teeth are the least capable of repair—practically, indeed, of none. Those tissues which are less capable of repair or renewal are those most permanently and most obviously affected by malnutrition—witness the permanent bone-lesions of bad cases of rickets.

The dentine or enamel of a tooth, once formed, can never be repaired or renewed; it is there for life, unchangeable except by destruction; and this is especially true of enamel. Hence that part formed during illness, or malnutrition, remains ill-formed as a permanent record of that illness; or the malnutrition may be so acute that no tissue (enamel is in our minds) at all is formed. Hence defects in shape, size, and soundness of tissue may all be looked to in the tooth as a record of former depression of vitality of the whole organism.

DENTAL AID FOR THE CHILDREN OF THE POOR.

The possibility of organising dental aid for children of the poor on a large scale is a question worth serious attention. An enquiry made some ten years ago by a committee of the British Dental Association, involving an examination of the mouths of 10,500 English and Scotch children of an average age of twelve years, showed among these children 37,000 unsound teeth—18,000 temporary teeth, more than half of which could, and should, have been "stopped;" and 19,000 permanent teeth, 13,000 of which should have been "stopped." These were of the Board-school class: the results were worse in children of a higher class.

The field for work is enormous, and the difficulties correspondingly great. Chief among these must always be the difficulty which public bodies will find in sufficiently remunerating a competent dentist to make it worth his while undertaking the work. To the School Dentists' Society one naturally looks for some practical suggestions in this field. Mr. J. W. Parr (*Trans. of the School Dentists' Society*, Vol. II., No. 9, 1902, p. 7) records his experiences as a salaried dental surgeon to the North-Eastern Hospital for Sick Children. In 1900 he was able to attend 640 children, and to put in 230 temporary fillings and 273 permanent. Although he confesses that, owing to the small average attendance of each patient, little can be shown to prove the effects of the treatment, it seems to us from the whole tenor of his report that a great deal can be done in this direction by organised public bodies.

While on the subject of the increase of dental caries, we should like to echo an appeal made by Dr. J. Kingston Barton in the *British Dental Journal* (May 15, 1903). Wishing to see the effect on the permanent teeth of bad feeding in infancy, he tried to get definite information from the state of badly-fed calves, but soon found it was necessary to keep them under observation till full-grown (four years) before such observations could be made. He says: "If any dental or medical practitioners interested in this subject are aware of any really poor farmers who rear their calves on separated milk with calf meal, and who keep such beasts (female most likely) as future dairy cows, I should much like observations made on the lower front teeth, especially with regard to the absence of enamel, particularly in horizontal lines, as is so often seen in the human subject." There must be many practitioners able and willing to make such observations, and we hope that they will forward reports of any cases which they may have the opportunity of observing to Dr. Barton, as the investigation is one of considerable interest and importance.[1]

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[1] We shall be happy to forward to Dr. Barton any information sent to us upon this subject, if any of our readers can assist in this research and prefer to adopt this channel of communication.—Ed.

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TWO CASES WITH COMMENTS.

I.

A CASE OF INTRATHORACIC TUMOUR.

BY RICHARD ALCOCK, M.D. (VICT.).

THE following case has appeared to me to be worth recording, mainly, perhaps, on account of the difficulty experienced in arriving at a diagnosis. This alone, however, is apt to be the rule rather than the exception in cases of intrathoracic cancer; Hughes Bennett in his "Clinical Lectures" (Third Edition, 1859) records nine cases of cancer of the lung "in none (of which) did any combination or succession of signs exist which could induce anyone to pronounce that pulmonary cancer was present." The same author noted, in more than one of these cases, that there was "leucocythæmia." We, of course, to-day should designate the blood-condition "leucocytosis," not leucocythæmia; nevertheless in my case a careful blood-count led, for a short time, to a diagnosis of spleno-medullary leucocythæmia; and I venture to think that, under the circumstances existing at that time, this diagnosis was unavoidable. The further course of the case proved that this conclusion was wrong; but that it should have been arrived at at all seems, for several reasons, worthy of being placed on record.

Unfortunately no post-mortem examination was permitted; but I think that the symptoms and signs as they developed, and which I have tried to record in the succession in which they were observed, leaves no ground for doubting at least the main diagnosis—"Intrathoracic tumour, almost certainly cancer."

Mrs. X., aged 54, married, the wife of a tenant-farmer, lived in a low-lying village in Yorkshire, on the banks of the tidal part of the River Ouse. She was born at Todmorden, but has lived in this village for more than thirty years—for practically the whole of her married life. She had been strictly temperate in her habits, and had performed the ordinary duties of her station without difficulty.

Previous History.—As a child, it was feared that Mrs. X. was consumptive; but whether there was any ground for this beyond a bad family history it is now impossible to say.

At the age of 17 she consulted a physician for an enlargement of the thyroid gland; this caused annoyance solely on account of the disfigurement it produced. She is quite sure that she was, at that time, in no way ill or out of sorts; in particular, she states definitely that there was no palpitation of the heart; there was no staring appearance of the eyes to excite comment in others or to be noticeable to herself; and finally, though this must be taken only for what it is worth, as being in answer to a question to which her attention had not previously been directed, there was no thrill or pulsation in the swelling. Some ointment was prescribed, and the growth underwent some diminution; it never, however, disappeared entirely, but remained as a noticeable enlargement up to the present time. An account of its present size and appearance is given later. It had never been any inconvenience. Beyond this she had never required a doctor for any illness. She had borne five children, all of whom are now alive and well. She had an adherent placenta at her first confinement; this was "taken away" by Dr. Malcolm Morris, who was in attendance. Shortly thereafter, there was an alarming hæmorrhage, from the effects of which she took long to recover. Otherwise her labours were all normal, and she fed the four younger children at the breast.

Her menstrual functions used to be performed regularly and normally, but the loss was always somewhat excessive. The menopause took place about the age of 45 quite uneventfully. For the last few years she had suffered from some amount of cough and shortness of breath in the winter.

Family History.—Her father, a paternal uncle, and three elder sisters all died of consumption. Her mother, æt. 79, was alive and well, as was also a sister a year or two younger than herself.

History of the Present Illness.—On March 27, 1903, I was called to see Mrs. X. for the first time. She had then been troubled for a month or two with a bad cough, breathlessness, and palpitation of the heart; this was getting worse, and she was now so weak that she had to give up her household duties entirely.

I propose to defer any regular account of her "Present Condition" till we bring the history down to the month of June. For the present it is sufficient to state that there was no rise of temperature; the pulse was rapid and very weak, irregular in strength, some beats being barely perceptible and others dropped entirely; the lungs were resonant on percussion, and some wheezy rhonchi were audible on auscultation. She had not been confined to bed, and except for the chest-conditions and the feeling of weakness and inability to work, did not feel really ill. Her appetite was fair, but there was some amount of flatulence after meals and a tendency to constipation. The cough was hard and dry in character, and "shook" the patient considerably, a prolonged attack of coughing bringing up only a small amount of expectoration, which was a clear transparent fluid, with a little froth on the top, as it settled in the vessel. The case was looked upon as one of chronic bronchial catarrh with weak, embarrassed heart. She was ordered a stimulant-expectorant mixture and a pill of aloes and asafœtida. Considerable improvement was soon noticed in the cough and breathlessness, but the general weakness remained unaltered; the appetite fell off somewhat, and fulness and flatulence after meals became the most prominent and troublesome symptoms. A tonic regimen was adopted, and gradually some strength and appetite was regained; so that, at the end of April, she was able to sit out of doors in the garden during a short spell of warm weather.

On the evening of May 1, I called to see her, and found her in bed, with severe pains all over, especially in her back and head, with a temperature of 102°, suffering from an almost incessant cough, and feeling very ill indeed. This change for the worse was attributed to a chill contracted in the garden the day before, caused by a change in the weather. Influenza being prevalent at the time, the present troubles were diagnosed as being due to that complaint; and she was ordered an effervescing mixture of quinine and ammonia. The temperature came down to normal in a day or two, but the general condition had become considerably worse than before this attack; the weakness and prostration were much more pronounced; the attacks of coughing became very severe, and chiefly occurred as nocturnal paroxysms of considerable duration, interfering with her rest. The pulse was almost imperceptible at the wrist. The urine was almost totally suppressed, a very few ounces only being passed in the 24 hours. Sleeplessness too, apart from that caused by the paroxysms of coughing, was complained of every night. In short, she was reduced to a condition of mind and body truly deplorable. Treatment was directed towards stimulating and strengthening the heart's action, whilst a soothing linctus was kept at hand to control the attacks of cough when they occurred. After a few weeks' stay in bed, improvement slowly took place, but only to a limited extent. In due course she began to be able to lie on a couch for a short time, and later to sit in a chair.

She now noticed that her body was enlarged, so that she could not get her clothes to meet as formerly. On examination there seemed no reason for this beyond flatulent distension of the bowels and a relaxed condition of the abdominal muscles. Now also, for the first time, the ankles became œdematous, and later the legs, and even the thighs were similarly affected. The urine was still very scanty, and the heart's action exceedingly feeble.

On June 3 acute gastric troubles set in; vomiting began early in the morning and persisted incessantly, causing the greatest alarm to the attendants, and distress to the patient. Appropriate symptomatic treatment, however, controlled the vomiting; on June 4 she vomited once, and once only, and this closed the incident.

After the first, the bowels have been regular throughout the illness; there had never been any tendency to diarrhœa before.

The condition of affairs at this time was unsatisfactory; the weakness continued; the nights were bad; paroxysms of cough were frequent, especially at night; the appetite was variable, and required tempting; but, above all, there was the uncomfortable feeling that no progress was being made. The temperature was normal whenever it was taken, night or morning; but the utmost exertion of which Mrs. X. was capable was to move from her bed to her chair, and in the evening back from her chair to bed; and even this was rendered more difficult on account of the dropsical condition of the legs.

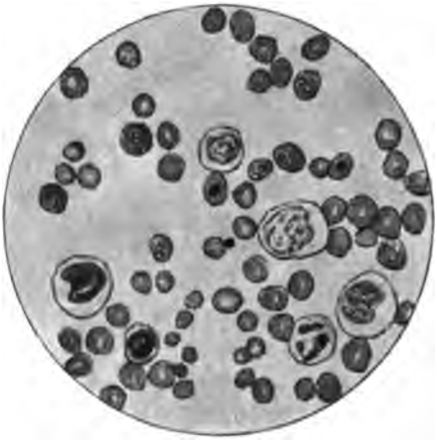
On Thursday, June 18, in the hope that it might throw further light on a puzzling condition, I prepared a stained blood-film, which, on examination, showed possibly a slight increase of leucocytes; but—what was more instructive—of the leucocytes a great number were very large uninuclear forms; and often with a moderate power ($\frac{1}{6}''$), a field could be found containing 20 leucocytes, of which perhaps 5 would be large uninuclear cells. Further search revealed the presence of nucleated red corpuscles (normoblasts). The information thus obtained gave strong ground for suspecting spleno-medullary leucocythæmia. There was no means of distinguishing large lymphocytes from myelocytes, as the preparation was stained with hæmatoxylin and eosine.

Present Condition (June 20, 1903).—The patient is somewhat emaciated; she has always been of slight build, but has become thinner since her illness began. The complexion is sallow, the lips rather blue, and over each cheekbone is a well-defined patch of dilated venules about the size of a half-crown. The expression appears somewhat drawn and anxious. The eyes are sunken as part of the general emaciation (there is no hint of exophthalmos, no von Gräfe's sign, no Stellwag's sign). The thyroid gland is enlarged symmetrically, without any thrill or pulsation. From side to side, across the front of the neck, it measures some 5 inches; vertically, each lobe measures some 2½ inches; the isthmus is well defined; the lobes seem to be about an inch in thickness in the thickest part; the whole mass, therefore, forms a flattened prominence.

Chest.—The respirations number 30 a minute: the breathing is rapid but unobstructed. The lungs are resonant, and ordinarily no râle or rhonchus is to be heard, except when the coughing paroxysms are about to come on. Expectoration then consists of clear, frothy, transparent mucous liquid, showing microscopically few formed elements (a few squamous cells, &c.). No tubercle-bacilli were present.

The heart was not increased in size; the sounds were feeble, but without murmur; the pulse-rate was 134. On this date it was impossible to count the pulse at the wrist, as many of the beats were so weak as to be almost imperceptible; and some

PLATE XXII.



A composite drawing of individual cells in a blood-film. (Mrs. X.) × 530. *Drawn with Zeiss prism. Showing myelocytes of various sizes, a lymphocyte and a polymorphonuclear cell; also a free nucleus. Among the red corpuscles great variety in the sizes is shown, and in some a pronounced vacuolation; also nucleated red corpuscles.*

were intermitted altogether. The heart's rhythm was regular, and under the present conditions of no work, palpitation is not complained about.

Blood.—The colour of the blood is abnormal, being duller in hue and darker in colour than usual.

Red corpuscles	- -	4,312,000 to cub. mm.
White "	- -	11,000 " " "

The differential leucocyte-count gave the following results:—

Small lymphocytes	- - -	8 per cent.
Large uninuclear cells	- - -	22 "
Polymorpho-nuclear cells	- -	68 "
Cells of undetermined character, degenerating	- - - - -	1·3 "
Coarsely-granular eosinophile cells	-	·7 "

Of the red corpuscles many were nucleated, some were vacuolated, many were so small as to be truly microcytes, and all took the eosine stain very badly, and this not on account of insufficient fixing. In some films, stained for the ordinary time, the red cells hardly appeared coloured at all. In the figure I have traced with a drawing-prism examples of these abnormalities (see plate). The drawing does not represent an actual field, but it represents actual cells, the outlines of which were traced under identical conditions, from one preparation.

The *Abdomen* was somewhat enlarged, but lax and flabby. There was no ascites. The spleen was moderately enlarged, projecting some inch beyond the costal margin. The liver was enlarged slightly; there was dulness in the right mammillary line from the sixth rib down to a point some inch and a half below the costal margin.

The *Urine* was scanty, depositing abundant amorphous urates; acid; S.G. 1022. It showed the very slightest recognisable haze of albumen on boiling; no sugar was present.

The *Legs* were œdematous, soft, pitting easily on pressure; the swelling of the legs disappears during the night, there being no trace of it in the morning.

Diagnosis.—The only points of a definite character so far observed were that the spleen was moderately enlarged, as was the liver; that there was some excess of leucocytes, and of these a considerable number were myelocytes or at any rate very large uninuclear forms; that the red corpuscles were degenerate, and some of them nucleated. Repeated examinations of the chest had only yielded negative results. The weak heart, the general muscular debility, the œdematous legs, in conjunction with the above more definite observations, led me to consider the case an early stage of leucocythæmia. But even at that time several difficulties were apparent; the blood was *dark* in colour (as taken from the lobe of the ear); there was almost an absence of eosinophile cells; and, chiefly, the general condition was very much more unsatisfactory than one would expect with so slight a degree of leucocythæmia and so moderate an enlargement of the spleen. Nevertheless, this diagnosis was the best available for a fortnight.

Progress (July 3).—The left hand was said to have been swollen the day before, so that she could not wear her wedding-ring, but no œdema was observable to-day. During the last few days the patch of dilated venules on the right cheek had disappeared, whilst that on the left was more marked than ever. The cough was more troublesome, and the general appearance was more distinctly anæmic.

A fresh blood-count gave the following results:—

Red corpuscles -	3,732,750, or 88·8 per cent.
Leucocytes - -	5,131

Hæmoglobin (by Gower's hæmoglobinometer against a white cloud) 80 per cent. In the fresh blood a number of poikilocytes observed.

Differential leucocyte-count:—

Small lymphocytes - -	17	per cent.
Large „ - - -	12	„
Polymorpho-nuclear - -	69	„
Eosinophile cells - - -	2	„

This blood was collected in the afternoon during digestion, the former sample early in the morning.

July 7, 1903.—The left side of the chest is decidedly smaller than the right, and moves less in respiration. On percussion, both sides are resonant, the right perhaps hyper-resonant. On auscultation, the breath-sounds are vesicular and normal over the whole of the right pulmonary area; on the left side the breath-sounds are markedly harsh. On measurement at the same level, the circumference of the left half is 14 inches, and the right 15½. *Pain* is elicited on percussion or firm pressure below and outside the angle of the scapula on the left side. Examination of vocal resonance and vocal fremitus gave no information, both being apparently normal.

For some time cough had come on in paroxysms on lying down ; now there is almost absolute orthopnœa, but the patient had to recline rolled over somewhat on to the right side.

Observations directed towards the condition of the recurrent laryngeal and sympathetic nerves gave negative results.

The left radial pulse was fuller and larger than the right. The left hand is now distinctly swollen. The colours of the two hands, placed side by side, are in marked contrast ; the right being yellowish, pale, and waxen ; the left, by contrast, distinctly bluish. The face, too, showed the same difference on the two sides, both as to colour and fulness, the right side being pale and shrunken, the left fuller and blue with the prominent patch of dark dilated venules on the cheek. Further, the large veins at the root of the neck were dilated on the left side, inconspicuous on the right. There were no enlarged glands to be felt in the axilla or in the supra- or infra-clavicular regions. Emaciation was becoming more marked, and the general debility decidedly increasing.

At the first blood-examination a plentiful supply was obtained from the right ear. At the second no sample was obtainable from the right ear or from a finger on the right hand, whereas a copious supply was obtainable from the left ear.

July 14.—Vomiting had several times occurred, but she could still eat ordinary food, which is generally retained satisfactorily. The left hand was very much swollen indeed ; and the right hand now pitted slightly on pressure for the first time.

On the 17th, sight was becoming impaired, and hallucinations were frequent, the patient seeing imaginary people in the room. The mind was rather wandering. Last night a sharp attack of diarrhœa occurred, which was soon controlled.

On the 18th, at 4 a.m., she was obviously *in extremis*, but she lingered on till 4 a.m. July 19, when she died exhausted.

REMARKS.

This case was one of extreme interest to the clinical observer. It commenced apparently as an ordinary winter cough to which the patient was subject, but even in the early stages anomalous signs were present which ought to have given more information than they did. It was noticed even at the first that there was some degree of cyanosis and dilated venules, along with marked air-hunger; but physical examination of the heart and lungs gave no clue to the real condition. From the first, the enlarged thyroid gland was conspicuous; but as it had lasted without inconvenience for the whole of the adult life, I do not know even now what importance to attach to it. Then came the attack of influenza—if it was influenza. It had every appearance of the disease. After that, the profound constitutional disturbance led to a blood-examination, which, instead of being a help, led one astray, but called attention to the facts that the spleen and liver were enlarged.

The key to the situation was found on July 3, when the patient told me that she had had to remove her wedding-ring the day before. The interpretation of the pressure-signs I take to be that at first there was some venous obstruction on both sides of the head and neck—witness the cyanosis and dilated venules; that afterwards, on the left side, venous obstruction predominated, whilst on the right there was, in addition, arterial obstruction—probably of the innominate artery, as witnessed by the pallor of that side and the difficulty of obtaining a blood-sample. The left lung was probably the seat of secondary nodules, as evidenced by the tenderness of that side of the chest, the harsh breath-sounds, and the diminished capacity and movement.

The results of the blood-examination now took on a different meaning: the degeneration of the elements implied malignant disease without a doubt; though why there should be a large excess of myelocytes, I am at a loss to explain.

At no time was a solid growth to be made out by percussion either on the back or front of the chest.

Postscript.—Since writing the above, I find that Da Costa (*Clinical Hematology*, 1902) says, writing of differential leucocyte-counts in carcinoma:—"Relatively high percentages of lymphocytes, especially of the large variety, have occasionally been observed. . . . Myelocytes are extremely common."

II.

TETANY OCCURRING IN A PATIENT SUFFERING FROM GASTRIC CANCER.

BY JAMES BURNET, M.A., M.B., M.R.C.P. (EDIN.),

Clinical Tutor, Extramural Wards, Royal Infirmary; and Physician to the Marshall Street Dispensary, Edinburgh.

ALTHOUGH, as pointed out by Moynihan,[1] the occurrence of tetany in cases of gastric dilatation is not so rare as the number of recorded instances would lead one to suppose, still I do not think its association with malignant disease is at all common. At all events, the records of this complication of gastric cancer are sufficiently scanty to warrant me in giving a few particulars of a case which was recently under my care.

The patient was a woman, aged 50, married, with four children. In 1899 she had the operation of hysterectomy performed for malignant disease of the cervix uteri. Shortly afterwards she began to suffer from hæmatemesis and the other symptoms of gastric ulcer. This was treated by her medical attendant as a case of simple ulcer, and the patient made a fairly good recovery. In 1901 she came under my observation. She was then the subject of hæmatemesis and severe epigastric pain. The vomited material contained lactic and butyric acids, but no free hydrochloric acid. Under somewhat prolonged treatment she again recovered, but after this she was never able to partake of ordinary food. Her nourishment now consisted of milk-puddings, rusks, weak tea, and a little fish, the last-mentioned being only taken occasionally as a special treat. She remained comparatively well until the spring of 1902. She then had a recurrence of her former symptoms, and at this date I found on examination that the stomach was drawn over towards the right side, while the liver was enlarged and tender. Her agonising pain necessitated the use of hypodermic injections of morphia.

From this date onwards the patient was unable to leave her bed. She was now considerably thinner than before, and very anæmic. The bowels became obstinately constipated, requiring to be moved by hot-water enemata every other day. She had moreover, still further to restrict her diet, as she was quite unable to take fish, while after a time even milk-puddings had to be abandoned. Her main support was milk considerably diluted and occasionally home-made beef-tea or chicken-broth. She remained free from pain, and only vomited at rare intervals. I was sent for at the end of June as the patient was vomiting and had severe pain over the bladder. The urine for the last few days had been very dark in colour, and when it was being passed the patient experienced very great pain. Examination of a sample proved it to contain blood. As regards the stomach there was little change, save that it now seemed to be bound down at its lower part, while the liver was greatly enlarged and massive to the feel. Treatment was at once directed to the urinary condition, and within a week the blood had entirely disappeared, and the patient only complained of pain over the bladder when she moved. Nothing was retained by the stomach save dessertspoonfuls of skimmed milk. Towards the middle of July even this was vomited, and it was now found necessary to precede each feeding by the administration of 10 minims of liq. morph. hydrochlor. This enabled her, to some extent, to retain the milk, and she began to improve slightly. During my absence on holiday, however, she had a severe attack of hæmatemesis. On account of the severe pain she was given a hypodermic injection of morphia every evening.

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[1] "A Note on Gastric Tetany," THE PRACTITIONER, March, 1903.

After my return I saw the patient on August 21. She was still retching and vomiting, although all that she was having was an occasional dessertspoonful of peptonised milk, preceded by 15-minim doses of liq. morph. hydrochlor. At intervals she had teaspoonfuls of sherry in iced water. The tongue at this time was very dry and brown. At bedtime she was given a suppository of morphia (gr. ss.). On the 23rd the bowels moved of their own accord, although she had not had a natural motion for eighteen months. This occurred again, to my surprise, on the 25th. These two motions were fairly well formed. She continued to have a suppository each night, as she no longer rested comfortably without one. Every dessertspoonful of milk was likewise still preceded by a dose of morphia. The retching and vomiting were now much less frequent, and it was hoped that she would soon recover again, at least for a time.

On August 27 I saw her at 11 a.m. She had had a morphia-suppository as usual the night before, but it did not seem to have the same soothing effect, as the patient was very restless. I found her lying on her back with eyes closed and mouth wide open. The breathing was very shallow and somewhat irregular. The left hand was flexed at the metacarpo-phalangeal joints, but the terminal phalanges were extended. The thumb was strongly adducted and flexed, so that it pressed into the palm. The right hand showed flexion at the metacarpo-phalangeal joints, but the fingers as a whole were more extended than on the left side, while the thumb was closely bent into the palm. On both sides there was a well-marked bluish-black discoloration over the dorsum of the fingers, extending for fully one inch above and below the first inter-phalangeal joints, and slight pressure over these areas produced evident pain. The wrists were strongly flexed, and the elbows also, though to a much less marked extent. The feet were extended and the toes adducted. The contractures were continuous, the affected parts being stiff and rigid, any attempt at movement causing the patient to moan. The palms of the hands were moist, while the rest of the skin was dry and harsh. The pulse was very feeble and rapid. The body-temperature was normal. The patient was unable to speak or even to recognise any one in the room. When spoken to, she made no sign of recognition, though once or twice, when her lips were moistened, she opened her eyes slightly. Earlier in the day she had been retching, and it was said that her limbs became rigid immediately afterwards, but whether this was really the case or not cannot be positively stated. Her condition remained absolutely unchanged, and she died quietly at 3 p.m., without regaining consciousness.

One or two points in the history of this case call for comment. It will be noted that on the 23rd and 25th of August the bowels moved of their own accord, although they had not done so for eighteen months previously. This phenomenon I attribute to the large and frequently repeated doses of morphia which the patient was having. Constipation is produced by small doses of morphia given occasionally, but I am inclined to think that larger and more frequent doses may increase peristalsis by paralysing the splanchnic inhibitory nerves going to the intestine. Again, the discoloration over the finger-joints is somewhat unusual. Probably this may have been due to trophic changes in the parts produced by loss of nerve-tone.

Lastly, the occurrence of tetany has to be considered. In this connection I wish to point out three important facts :—

(i.) The case was one involving both stomach and liver, the disease in both being far advanced.

(ii.) The patient was taking a considerable amount of morphia daily.

(iii.) There was the history of a family quarrel some three days before which upset the patient very much.

My own impression is that the last-mentioned was the real cause of the condition. Otherwise, why should tetany not have shown itself before in the course of the patient's long illness? Again, the administration of morphia did not prevent its onset. At the time the patient was well under the influence of this drug ; in fact, life would have been a prolonged agony without it. It is not my intention at present to discuss the varied and manifold theories advanced as to the true origin of tetany in gastric disorders. Many of them, I think, are unnecessarily elaborate. If we bear in mind the close relationship of the nervous system to the organs of alimentation we shall not have much difficulty in explaining why tetany should result in cases where the stomach is irritated, as it undoubtedly is when it is dilated or is the seat of malignant disease. My chief object in publishing this case is to induce others to do likewise, as I am firmly convinced that gastric tetany, even in malignant disease, may not be so uncommon as is generally supposed.

Public Health.

SUMMER DIARRHŒAL MORTALITY IN LONDON.

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ANY inquiry into summer diarrhœal mortality in London must take account of the deaths registered as due to enteritis and gastro-enteritis, as well as of those referred simply to diarrhœa and cholera. In a short paper by the author, published in the *British Medical Journal* of May 28, 1898, attention was drawn to the significance of enteritis in the metropolitan returns, and reasons were given for the view that a very large majority of the deaths registered as due to enteritis should be transferred to the "diarrhœa" column. The average seasonal mortality-curve of enteritis in London was shown to be almost identical with that of diarrhœa, the almost simultaneous rise of the two curves at the end of June, the simultaneous attainment of the maximum of each at the end of July, and the rapid fall of each curve throughout August and September presenting features of almost complete harmony throughout their course. Taking the 12 years 1891–1902, 77 per cent. of the deaths from diarrhœa and cholera have occurred, on an average, in the third quarter of the year, and 60 per cent. of the deaths from enteritis have so occurred. Again, the age-incidence of diarrhœa and enteritis on the population is very similar. In the same period (1891–1902) 91 per cent. of the deaths from diarrhœa and cholera have been of children under five years of age, whilst 78 per cent. of the deaths from enteritis have been under five.

In each of the years 1891, 1892, and 1893, 30 deaths from enteritis and gastro-enteritis were registered for every 100 deaths from diarrhœa and cholera. In 1894 there were 51 due to enteritis to every 100 due to diarrhœa; in 1895, 40; in 1896, 51; in 1897, 52; in 1898, 55; in 1899, 73; in 1900, 58; in 1901, 36; and in 1902, 37. For the first three quarters of 1903 there have been 28 deaths from enteritis registered for every 100 due to diarrhœa and cholera. It is evident, then, that from 1893 to 1899 "enteritis" registration was increasing; since 1899 there has been a decrease in the use of the term enteritis as a cause of death. This decrease dates from the action of the Superintendent of Statistics in the Registrar-General's office in calling attention to the unsuitability of the terms enteritis and gastro-enteritis for the registration of deaths from what is in reality summer diarrhœa, and suggesting the use of other terms, more especially "zymotic enteritis," which now enable these deaths to be recorded under the "diarrhœa" column.

In the following table, which shows the relations subsisting in each of the years 1891–1903 between diarrhœal mortality in London in the summer months, and the average temperatures and rainfalls of those months, deaths from enteritis and gastro-enteritis have been included with those due to diarrhœa and cholera. Some of the deaths from enteritis of persons over five years of age are probably due to disease essentially different from summer diarrhœa; but these form so small a proportion of the whole in the summer months that there is much less error to be feared from their inclusion than would arise from the exclusion of enteritis in a general survey of summer diarrhœal mortality.

An examination of the table shows that the June mortality from diarrhœal diseases in London is not heavy. The sudden upward rise in the seasonal mortality-curve does not, as a rule, take place until the 25th or 26th week of the year, that is to say, in the last week or ten days of June. The mean temperature of this month (average of 1891–1903) is 59·6° F. The year 1893 is seen to be exceptional. In this year the curve began to rise at the end of May or very early in June, and the mortality for the month of June was more than three times in excess of the average. The May of 1893 was warmer than any May of the thirteen years (1891–1903), the mean temperature being 57·7° as against an average of 52·8°, and the rain-fall of 0·53 inch was also very deficient. The June of 1893 was also warm (mean temperature 61·8°), and its rain-fall 0·82 inch—a small one. The warm and dry months of May and June in 1893 corresponded with an unusually early rise in the summer diarrhœal mortality in that year.

DEATHS IN LONDON FROM DIARRHŒA AND ENTERITIS, 1891–1903.

Year.	Deaths.					Mean Temperatures.						Rain-fall.						
	June.	July and August.	September.	October.	Five Months, June to October.	May.	June.	July.	August.	September.	October.	May.	June.	July.	August.	September.	October	Total Six Months.
1891	138	1,709	394	256	2,497	50·3	60·4	60·3	58·6	59·1	50·9	2·69	0·96	3·39	3·72	0·82	4·32	15·90
1892	228	1,678	482	178	2,566	55·2	58·1	59·6	61·7	56·3	45·4	1·66	2·27	1·54	3·03	2·01	3·88	14·39
1893	729	2,252	592	242	3,815	57·7	61·8	63·1	65·5	57·1	51·5	0·53	0·82	3·33	1·25	1·29	4·16	11·38
1894	96	1,349	348	169	1,962	50·3	58·6	62·0	59·6	54·1	50·2	1·52	2·04	3·26	3·03	1·25	3·99	15·09
1895	247	2,966	624	418	4,255	56·0	61·4	62·6	62·2	62·2	46·5	0·45	0·21	3·89	2·14	0·93	2·69	10·31
1896	277	3,403	360	191	4,231	54·8	63·4	65·2	59·2	56·7	46·5	0·27	1·94	1·07	2·06	5·54	2·80	13·68
1897	176	4,660	510	201	5.547	52·3	61·1	64·5	62·9	55·2	51·0	1·25	1·93	0·73	2·86	2·70	0·48	9·95
1898	135	3,185	2,112	606	6,038	51·7	57·6	61·5	64·6	62·1	53·8	2·64	1·75	1·34	0·86	0·31	3·15	10·05
1899	150	4,699	1,390	326	6 565	50·9	60·7	65·7	65·7	57·7	49·1	1·65	0·76	1·74	0·35	2·23	2·34	9·07
1900	146	3,097	1,013	450	4,706	51·6	59·5	66·8	60·6	57·9	51·1	1·37	2·82	1·41	2·03	1·14	1·55	10·32
1901	163	3,055	875	345	4,438	53·2	58·8	64·8	62·3	57·9	50·5	1·79	1·49	1·72	2·03	1·35	2·60	10·98
1902	95	897	1,110	470	2,572	48·4	57·6	61·1	60·6	56·6	49·8	3·33	3·10	1·09	2·93	1·65	1·24	13·34
1903	133	1,246	837	507	2,723	53·6	55·8	62·1	60·1	56·6	51·9	1·95	6·07	5·27	5·66	1·39	4·43	24·77
Average 1891–1903	209	2,630	819	335	3,993	52·8	59·6	63·0	61·8	57·7	49·9	1·62	2·01	2·29	2·46	1·74	2·89	13·02

The years 1894 and 1902 were characterised by cold Mays and Junes, and their diarrhœal mortalities were much below the average. The years 1895 and 1896 had warm Mays and Junes, and in 1895 the rain-fall of these two months was exceptionally small. In each of these years the June diarrhœal mortality was slightly over the average, but nothing approaching the mortality of June 1893. It is evident, therefore, that, whilst a warm May and June tend to produce an early rise in the diarrhœal mortality-curve, there is no such direct correspondence as would lead to the belief that a high air-temperature in these months is the only factor concerned in initiating a high prevalence of diarrhœa.

The mortality-figures for July and August have been taken together, as it is impossible to separate out in the Registrar-General's returns the exact figures for each month. They really represent the mortalities accruing in the 27th to the 35th weeks of the year.

The years of maximum mortality in July and August (1897 and 1899) are characterised by mean temperatures in July and August above the average. But here again there is no proportional correspondence between high temperature and heavy mortality, otherwise the mortality in July and August, 1893, should much more nearly correspond with those of 1897 and 1899 than it does. As a matter of fact, it was less than half that of either of these two years; but the temperature-records of July and August, 1893, are very similar to those of 1897 and 1899; and on inspection of these figures alone one might have anticipated a mortality in July and August, 1893, not far short of those displayed in July and August of either 1897 or 1899. The year 1900 showed an exceptional behaviour of diarrhœa-prevalence. The July temperature of 66·8° F. is the highest recorded for the series of years; but the July mortality was low until the fourth week of the month. A heavy mortality prevailed throughout August and September, notwithstanding that these months were comparatively cool. In 1902 the July and August mortality was only about one-third of the average of these months. In this year July and August were cold inclement months, but not apparently worse in this respect than the years 1891 and 1892, when the July and August mortalities were about double that of 1902.

There is, therefore, some factor apart from temperature which exerts a very considerable influence on the seasonal prevalence of diarrhœa. This factor is apparently not rain-fall. A consideration of the rain-fall records for the summer months of 1891-1903 seems merely to suggest that rain-fall *per se* is not a disturbing factor, apart from the fact that high summer temperatures are usually noted in periods of deficient rain-fall. A fine, hot summer is usually a dry summer; but excess or deficiency of rain-fall, considered apart from temperature, does not appear to be influential in affecting diarrhœal prevalence and mortality. The diarrhœa-mortality of the third quarter of the present year, 1903, has been low, but it is slightly higher than the mortality for the corresponding period in 1902. The temperatures of the two seasons have been very similar, but whereas the rain-fall of the third quarter of 1902 was only 5·67 inches, that of 1903 was 12·32 inches. If excessive rain-fall tended to induce low diarrhœa-prevalence, then the diarrhœa of the present season ought to have been much below that of 1902.

The September mortality shows the decline of the seasonal prevalence of diarrhœa. Prior to 1898 the decline appears to have been a rapid one each year, with the exception of 1895. In this year the mean temperature of September (62·2° F.) equalled that of August, and high diarrhœa-prevalence continued well on into the fourth quarter of the year. In 1898, also, September (62·1°) was warmer than July (61·5°), and the diarrhœa-prevalence was relatively very high in September and October. In 1899, however, the September temperature was equal to the average, and the diarrhœa-mortality fell very sharply during this month, although the July and August mortality was the heaviest recorded. In 1900, in 1901, and in 1902, diarrhœa-prevalence was continued for some unexplained reason until the end of October, although the September temperatures hardly departed from the average. The year 1902 was very exceptional as regards the behaviour of diarrhœa. The September mortality was greater than that of July and August taken together, owing to the lateness of the onset of epidemic prevalence, but September was a cold month, the mean temperature being only 56·6°, as against an average of 57·7°.

As for the rise, so for the decline in diarrhœa-mortality, temperature appears to exert a certain influence, but there are other factors operating, which in certain seasons tend to mask its influence. What these other factors are is not at present known. Infectivity might be thought of as controlling the decline of an epidemic. If the disease is in any way infectious, then the existence of a very large number of infecting centres would tend to perpetuate the epidemic; and the decline, notwithstanding a lowered temperature, would be very much more gradual than it is. The facts hardly warrant the assumption of any high degree, at any rate, of infectivity; as, if this were so, the late autumn prevalence would be greatest and most prolonged in those years in which the summer prevalence has been highest. But this is not the case as the table clearly shows. It must be remembered, however, that we are only dealing with mortality and not with actual prevalence, as the cases that end in recovery are unrecorded; and it may possibly happen that different epidemics have different fatality-rates; and that prevalence is not always to be measured by mortality.

The substance that sets up summer diarrhœa is now generally believed to be introduced into the human body by means of the food. Of the London deaths from diarrhœa and cholera from 1891–1902, 91 per cent. were of children under five, and 77 per cent. were of infants under one year. Of the deaths due to enteritis and gastro-enteritis for a similar period, 78 per cent. were under five and 65 per cent. were under one year. The incidence of the disease, therefore, so far as its fatal effects are seen, is chiefly upon infants and young children. As milk forms the principal food at this early period of life, it would seem probable that it is in this fluid for the most part that the substance is conveyed to the alimentary tract, which ultimately sets up an irritative diarrhœa. The substance so present in milk, whether it be a bacillus with its toxines, or a ferment, is evidently largely dependent upon air-temperature and some other unknown factors, for its subsequent capacity to set up gastro-intestinal changes when introduced into the alimentary tract. In the absence of a sufficiently high air-temperature the poison, although present in the milk, appears to be inoperative. Whether this poison is present in the milk as supplied by the retail milk-vendor, or whether it is in the homes of the poor that the milk receives a contamination not heretofore present, are questions not altogether solved. What, however, we do see is that Nature by sending us warm and cold summer seasons demonstrates on a large scale that diarrhœa-mortality is very largely a matter of temperature; and our scientific reasoning enables us to realise that a process so largely dependent upon temperature must have relation with some organised ferment or bacterium whose habitat is food, this being the vehicle for the introduction of the *materies morbi* into the human body.

The most hopeful methods, therefore, of counteracting the diarrhœal poison would appear to be the sanitary control and supervision of all milk-supplies, and the education of the mothers of the children to an appreciation of the necessity for domestic cleanliness and for the careful and cleanly preparation of the children's food.

ANATOMICAL STUDY.

By "AN OLD TEACHER."

THE student of anatomy of the present day has things made very easy for him in comparison with his brother of fifty years ago. Not only are our museums filled with anatomical preparations of a perfection and beauty which render them a delight to the eye as well as to the mind, but the modern systems of anatomy are things of beauty compared with those of former days. The works of Gray, Morris, and Cunningham, to take them in their historical order, are handsome volumes, brought well up to date and profusely illustrated, and one of these will enable the student to learn his anatomy—so far as it can be learnt from books—with ease and celerity. Whether the anatomy learnt so easily sticks in the memory as well as that more arduously obtained in former years, may possibly be questioned—especially by examiners.

Erasmus Wilson's *Anatomist's Vade-mecum*, illustrated with woodcuts by Bagge, and published in 1840, was a favourite in its day, and was the pioneer of illustrated manuals of anatomy. *Quain's Anatomy* was also much read, and woodcuts were introduced into it; but these, like those in Wilson, were of small size, and of not much assistance to the student. Holden's *Osteology*, published in 1855, was really the first attempt to lighten the labours of the student by the graphic method, and his plates of the bones, with the origins of the muscles printed in red and the insertions in blue, opened up a new method of teaching, for each school was soon provided with a set of bones painted according to this pattern. Anyone who can remember what it was to try to learn his bones with the aid of Ward's *Osteology* only, will recognise the benefits Holden conferred on successive generations of students.

Gray's Anatomy appeared in 1858, and adopted the graphic method, not only for the bones, but for all the other tissues of the body. The attachments of muscles were marked only in black, but the names were printed on each bone, muscle, vessel, and nerve in a way which had not been attempted before. Naturally "Gray" proved a great success, and now we have the fifteenth edition, published in 1901 under the editorship of Mr. Pickering Pick and Professor Howden, with colours introduced into the original blocks, and numerous illustrations taken from preparations in the museum of the College of Surgeons and from various other sources. We must confess to being a little disappointed in the "process-blocks" from preparations in the Hunterian Museum, which do not bear comparison with the old woodcuts, and we regret to see Flower's plan of the fifth cranial nerve reproduced with the lenticular ganglion upside down! The strictly anatomical part of Gray comprises 1,055 pages, and it is preceded by an essay of 148 pages entitled "General Anatomy or (*sic*) Histology," in which a brief and unsatisfactory epitome of physiological anatomy, including development, is given, but is rather out of place.

The third edition of *Morris' Anatomy* was published in 1902, and is the work of several anatomists under Mr. Henry Morris' editorship. One of the authors, Mr. Arthur Hensman, died some time back, and we have quite lately had to lament the death of Mr. Walsham, whose article on the blood-vessels and lymphatics is one of the best in the book. Following closely the lines of Gray as regards the illustrations, of which 266 are coloured, 1,270 pages are devoted to the details of anatomy, and so far as we have noted, with the greatest accuracy. A section on Surgical and Topographical Anatomy by Mr. Jacobson is a valuable addendum to the purely anatomical details of the preceding sections, and contrasts favourably with the paragraphs on surgical anatomy scattered through Gray, with antiquated and often incorrect illustrations taken from Hind. Walsham's article is beautifully illustrated by coloured drawings from preparations in the museum of the College of Surgeons and of St. Bartholomew's Hospital, many of the latter being Mr. Walsham's own work. These are all woodcuts, and are admirably engraved and printed in colours, and we regret the more therefore that the name of the printer is nowhere given.

Cunningham's *Text-Book of Anatomy* is, like Morris', a compilation by various authors (all but one of whom have been pupils and assistants of Sir William Turner), edited by his successor in the chair of anatomy at Edinburgh. Like Gray, Professor Cunningham devotes the first sixty-six pages to an introduction to General Embryology, which we must confess seems to us out of place; then follow the bones, joints, muscles, nerves, vessels, &c., concluding with eighty-six pages of Surface and Surgical Anatomy by Mr. Harold Stiles. The illustrations are 824 in number, and original, as far as they can be, and reflect great credit on the artists, wood-engravers and printers, all of whom, it need hardly be said, hail from Scotland. There is a Scotch pawkiness too about some of the descriptions, and we note that the Latin equivalents are dragged in frequently, and unnecessarily as it seems to us. But the *fovea capitis femoris* reminds us that the "pit at the root of the great trochanter" was in the old days of Stanley and Lawrence the recipient of many more muscles than are now allotted to it. We can especially commend Fig. 162, giving a back view of the upper end of the femur, for the distinctness with which the insertions into the great trochanter are shown. When we come to the lower end of the femur, however, we are greatly surprised to find that the gastrocnemius appears to have moved from the back to the side of each condyle, and is drawn immediately above the popliteus, in Fig. 163! Coming down to that *crux*, the fibula, we are glad to find all three authorities making the shaft quadrilateral, instead of prismatic, as used to be taught before the sixties, but for a complicated description commend us to that of the shaft of the fibula (*corpus fibulæ*). The author, Professor Thomson, says rightly enough that "the most important point is to determine the position of the interosseous ridge," and then proceeds to describe everything but the ridge, which is always easily found by following up the rough triangle above the *articular* surface of the outer malleolus. Again, Professor Thomson calls the anterior surface the *flexor surface* and proceeds "from this arises the *extensor* communis digitorum, &c., which though extensors of the toes are also flexors of the ankle." If this statement is not calculated to confound the young student, we shall be surprised!

The Muscular system is described by Professor Hepburn, now of Cardiff, and is extremely well illustrated, the anatomy of the groin being particularly well done; and then follows the Nervous system by Professor Cunningham. This essay occupies 150 pages, and is worthy of its author's reputation. The anatomy and development of the spinal cord and brain are given in detail and with great clearness, and the essay is illustrated most elaborately. The anatomy of the nerves, spinal, cranial, and sympathetic, is by Professor Paterson, and the description lacks clearness in places. Thus the account of the brachial plexus is difficult to follow, with Latin names unnecessarily introduced, and with a diagram which is so complicated as to be unintelligible. Figs. 442 and 444, illustrating the distribution of cutaneous nerves on the front and back of the lower limb, are simply impossible to follow. The Organs of Sense are by Professor Howden.

Professors Young and Robinson have undertaken the section on the Vascular System, which is one of the best in the book. Here the authors have the advantage of the modern system of using formalin to harden the tissues; and Fig. 551, showing the posterior wall of the pericardium after removal of the heart, is a good illustration of the process. In illustrating the distribution of the cerebral arteries three colours are used with great effect, and Figs. 557 and 558 are capital examples of successful colour-printing. The Respiratory System is by Professor Cunningham, the Digestive System by Professor Birmingham, and the Urogenital System by Professor Dixon, of Dublin. *À propos* of the recent activity in the surgery of the prostate and the successful removal of the organ by Freyer and others, we have sought for confirmation or otherwise of the view laid down by Sir Henry Thompson nearly fifty years ago, viz.: that the prostate has a distinct capsule of its own apart from the capsule derived from the pelvic fascia. We find no confirmation of this important point in Cunningham or Morris; but Gray (p. 994) says, "The prostate is immediately enveloped by a thin but firm fibrous capsule, distinct from that derived from the recto-vesical fascia, and separated from it by a plexus of veins."

Having now briefly reviewed the three most modern systems of anatomy, let us for a moment consider whether these books and lectures arranged upon the same plan are, after all, the best means of teaching the future medical man the anatomy of the human body. It is very like the system of Chinese metaphysics pursued by Mr. Pott's literary drudge, for the student is told to get up bones, ligaments, muscles, vessels, and nerves separately, and then to "combine his information" on the living patient. Of course, it will be said that dissection teaches him to do this combination; but does it? In the old days, when a student dissected for three winters, and had to pass in anatomy at the same time as surgery, he went into practice with at least some knowledge of anatomical details; but now, when he has but two winters for dissection, and has to give up several hours a week to practical physiology, he does not dissect as he did formerly, but expects the demonstrator to do the greater part for him, besides giving him demonstrations and generally coaching him upon museum specimens. Then, the examination in anatomy and physiology once achieved, good-bye to all anatomical work until just before the final examination, when the teacher of practical surgery tries, and too often tries in vain, to recall the anatomical details which the candidate ought to have at his fingers' ends. Let us look at the return of the results of the Primary Examination for the Fellowship of the College of Surgeons, which is generally taken by young Members of the College within a year or two of qualifying; and what do we find? In November, 1902, there were 110 candidates, of whom only 38 passed; and in May, 1903, of 128 candidates only 49 passed; or of 238 candidates in the year only 87 were successful!

But undoubtedly a certain amount of dissection is still done in the schools, and for this purpose a dissecting book is needed, and here again we have three new works or new editions. Heath's *Practical Anatomy* has suffered many things of successive editors, but Mr. Ernest Lane, who edited a ninth edition in 1902, has done his best to reduce the bulk of the book without omitting essentials. The illustrations from Hirschfeld and Leveillé were the first reproductions of their beautiful *Névrologie* in this country, but have now become common to Quain, Morris, and Hughes, and the coloured plates from Maclise, which were omitted by Anderson in the eighth edition, have been replaced in their several regions. The book has stood the test of nearly forty years' existence, and we think it has still a career before it.

The *Practical Anatomy* by the late Professor Hughes of King's College is a more ambitious work, which unfortunately its author did not live to complete. It is more a systematic work on anatomy than a dissector's guide, for few, if any, directions for dissection are given, and the parts are not described in the order in which they would be met with in dissection. The work is in three volumes, and in the first, which alone had its author's superintendence, are a number of original lithographic plates drawn from preparations in Professor Hughes' museum. To draw the femoral artery twisted by injection, as it is in plates 22 and 23, is to give a very inaccurate idea of the position of the vessel in life; and plates 29 and 31 require a good deal of study to make out what part of the body they represent. A series of woodcuts of the bones with the attachment of muscles, by Professor H. E. Clark, are introduced, and numerous figures are borrowed from various sources. Dr. Keith, of the London Hospital, is responsible for the second and third parts, and the same faults exist there as in the first part, but no lithographic plates are inserted, the woodcuts being extensively borrowed for the most part, and the original drawings being very coarse in execution.

Professor Cunningham's *Manual of Practical Anatomy* has reached its third edition this year, appearing in two volumes. This in itself is an evil and raises the price of the work, but the beauty of the illustrations will no doubt compensate for other drawbacks. Coloured plates of the bones are introduced with the origins and insertions of muscles duly recorded. This cannot but be of great help to the beginner, who will, however, we hope, not fail to compare the figment with the reality. Many of the illustrations are borrowed from the author's *Text-Book of Anatomy*, and others have been provided by the artistic hand of Mr. J. P. Murray; they are mostly printed in colours, and reflect credit on all concerned in their production.

We are glad to find that Edinburgh does not agree with Oxford in changing the origin of the gastrocnemius, and that extensors of the toes are not said to rise from the flexor surface of the fibula. The abdomen is extremely well illustrated, and we rejoice to find the liver drawn, what we should call, the right way up. Fig. 152, showing the posterior surface of the liver, will help the novice to understand the formation of the coronary and lateral ligaments, and Fig. 159 shows admirably the relations of the kidney and pleura seen from behind.

Anent the prostate we find (page 531): "Within the capsule formed by pelvic fascia and subjacent to the veins a thin sheath forms an immediate investment for the prostate."

The second volume of the manual comprises the dissections of the thorax, and head and neck, and is also profusely illustrated.

The student of to-day, having no hospital attendance in his first two years, ought to be able to assimilate the two great subjects of anatomy and physiology satisfactorily. If he does not, upon whom are we to lay the blame, the teacher or the taught? We believe the teacher is sometimes to blame for teaching what is called "advanced" anatomy and physiology instead of the plain facts of both subjects which will be useful to the practitioner. We hear a good deal nowadays of these "advanced sciences," particularly in connection with the ambitious views of the University of London; but may we venture to suggest that we have to do with the average *practitioner* after all, and that if Cambridge and London will not condescend to teach the anatomy and physiology needed for practice, the Universities of Birmingham, Liverpool and Leeds, to say nothing of the Scotch Universities, will be prepared to step in and fill the gap? Already it is found that it "pays" to go to Edinburgh or Glasgow and come away at the end of five years with a degree in medicine, almost as a matter of course; but how will the London medical schools exist when the English provincial Universities enter upon a similar competition?

Post-mortem Examinations.

THE question of post-mortem examinations has been much to the front lately, owing to the action of one of the London coroners in appointing a pathologist to make all the autopsies necessary in his district, instead of getting the practitioner in attendance on each case to perform this duty. This course has not unnaturally caused a good deal of heart-burning among those who are thus practically declared unfit to make post-mortem examinations, and has raised the whole question in an acute form. We must confess that on abstract grounds we are in favour of skilled pathologists being employed for these duties, as being on the whole the most satisfactory arrangement, but we certainly do not hold that the appointment of such officers should be left to the discretion of the coroner. Many coroners possess legal and not medical qualifications for their offices, and cannot be expected to act as judges of the pathological acquirements of candidates for such a post. Further, we are most distinctly of opinion that the evidence of the medical man who has been in attendance on a patient—if such has been the case—should in all instances be heard at the inquest, since pathological data alone without clinical observations possess much smaller value. We have heard of a pathologist not only diagnosing, from what he found, that a patient had died of a lymphatic diathesis, but also expressing an opinion as to the possibility of recognising the condition *intra vitam*, and we should have liked to hear what the clinician had to say on the point.

What the final outcome of this controversy will be, as to the methods generally adopted by coroners in the matter of post-mortem examinations, we cannot of course prophesy, but we may use the occasion as a peg on which to hang the reflection that it would be well if more attention were paid to the making of autopsies by medical students during their period of training. The value of the knowledge thus obtained cannot be over-estimated. Nothing can so well impress on the mind the actual physical facts underlying diseases, in the clinical sense of the term, as the study of the morbid conditions found *post mortem* in a case which has been observed during life. Further, it may fall to the lot of any practitioner to have, at some time or other, to make an autopsy, and we do not altogether envy the position of one who has to do so for the first time, never having practised the art under guidance as a student.

As to the actual routine of a post-mortem examination, probably there is a good deal of divergence between the methods of different operators. A book has recently appeared in France from the pen of Dr. Letulle,[1] which offers a very full account of all the steps of the procedure, illustrating them with an abundance of figures to show the minutest details of manipulation. It is the fullest account of this branch of pathology with which we are acquainted. As the writer points out in his preface, "The current methods of procedure which students and practitioners see employed in the post-mortem theatre are of very varied origin. Some arise in the customary employment of a special trick (*tour de main*) learnt in the school of our predecessors; others are the product of purely empirical habit; others, finally, result from the study either of ancient articles buried in bygone periodicals, or of some French manual like that of Bourneville and Bricon or that of Suchard, or of a foreign treatise such as the 'Sektionstechnik' of Virchow, Orth's 'Manual' or Nauwerck's 'Précis.'" This is as true of this country as of France, for we doubt whether students of medicine ever read any books on the subject of making necropsies, and traditions are handed down in our various schools from pathologist to pathologist.

Dr. Letulle's work is a valuable compendium of the branch of science with which it deals. We may mention in illustration some of the advice which he gives and the rules which he lays down.

With regard to the actual post-mortem room and its fittings it is noteworthy that Dr. Letulle considers that the whole should be kept as aseptic as an operating-theatre, a condition of things that is far from being the case at more than one of our London hospitals. The room is to be fitted with taps that are turned by the foot, and all fluids that enter the drains are to be first sterilised. The table used is to be fixed, not rotatory, and special rules are laid down for its design. A full armamentarium of instruments is recommended, but among them we do not find mention of the useful "coronet" for the removal of the scull-cap, though it is certainly of considerable assistance in this operation. Gloves of any kind are not to be worn, except by those who have unduly delicate skins; they spoil the subtlety of the sense of touch, and are a source of false security—"evil counsellors."

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[1] *La Pratique des Autopsies*. Par M. LETULLE. Avec 136 dessins d'après nature par C. Regnier. Paris: C. Naud. 1903.

A careful inspection of the exterior of the body is first carried out, special attention being paid to the orifices. The usual longitudinal incision is then made, and the organs are examined as they lie *in situ*. The parts occupying the neck are next carefully dissected, the larynx, trachea and œsophagus being separated from the structures on which they lie, while the muscles are as far as possible preserved intact. Care is taken not to wound the great veins in the process, and to preserve the point of entry into them of the thoracic duct. The clavicles are removed with equal precautions, and the breast-piece raised, the ribs being preferably sawn through rather than their cartilages divided. If there is any question of the existence of a pneumothorax, a small piece of one of the intercostal muscles is carefully resected so as to leave the pleura intact beneath; the hollow thus made is filled with water, and a puncture is made through this, so that any bubbles of gas which escape may be readily observed.

Dr. Letulle directs that the whole of the viscera, thoracic and abdominal, are to be removed in one connected mass from the mouth to the anus. We do not feel sure that this procedure is to be recommended to anyone but an expert pathologist, as the beginner will find it much more difficult to recognise the relations of the parts when they have been removed than while they lie in their normal positions. Instances must be few in which such an entire evisceration *en masse* is absolutely needed, though it is often necessary to remove the thoracic organs entire, and occasionally the abdominal contents similarly. And here we may remark that the writer estimates the time necessary for an ordinary autopsy as at least three hours, which is more than it is possible to afford in very many cases, especially if more than one cadaver has to be examined.

We cannot follow our author in his ample description of the procedures to be adopted in the special examination of each organ in its turn. Nothing appears to be neglected in this treatise; and the figures so admirably show the exact lines of incision to be made, and the positions of the hands in the process, that even a beginner can without difficulty grasp most of the technique of the operations. Those who are interested in this branch of medical work cannot do better than procure the book and study it for themselves.

Books in Brief.

THE subject of tuberculosis and the open-air treatment is so much in evidence that we may expect books on it to be numerous. A good account of the disease and its diagnosis, prognosis, and treatment is given by Professor Bridge[1] in some recently-published lectures. "The cardinal doctrines," he says, "to be emphasised at all times are: Keep well and normally strong; always breathe the best and cleanest air; and avoid the bacilli of tuberculosis—not by making social pariahs of the sick, but by a never-ending campaign for the destruction of these microbes." A convenient account of the open-air treatment,[2] explaining its principles and the regulations for carrying it out to the best advantage, is to be found in a little book by Dr. Ransome. The influence of feeding, rest, and exercise is discussed, and problems, practical and theoretical, such as the disposal of sputum and the resistance offered by the system to tuberculosis, are dealt with briefly, but satisfactorily. Plans for a sanatorium are also given, and directions as to the essentials of such an institution. Those who are interested in the steps being taken to combat the disease abroad may consult with advantage a little handbook issued by Messrs. Naud of Paris on the Sanatoria in France.[3] The questions to which we have just alluded are discussed from the French point of view, and illustrations of the different institutions are given. Some rules for the guidance of tuberculous patients are supplied in a little brochure (French) of 20 pages from the pen of Dr. Savignac,[4] issued by the same publishing house, and advice on nursing a consumptive patient is offered to our own countrymen and countrywomen in an unpretentious little volume called "How to take Care of a Consumptive."[1] The hints contained in the latter appear to be useful and good. We quite agree with the concluding sentence that "much depends upon the care received not only from the medical man, but from the nurse and cheerful home surroundings." In this connection we may mention the recent volume of *Transactions of the American Climatological Association*,[2] which contains some studies on influenza and tuberculosis, as well as on health-resorts in the United States and Canada. The illustrations accompanying the articles are especially good.

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[1] *Tuberculosis.* By Norman Bridge, A.M., M.D., Emeritus Professor of Medicine in Rush Medical College. Philadelphia, New York, and London: Saunders and Co. 1903.

[2] *The Principles of the Open-Air Treatment of Phthisis and of Sanatorium Construction.* By Arthur Ransome, M.A., M.D. (Cantab.), F.R.C.P., Consulting Physician to the Manchester Hospital for Consumption, &c. London: Smith, Elder, and Co. 1903. Price 5s.

[3] *La Cure de la Tuberculose dans les Sanatoriums Français.* Par les docteurs Plicque et Verhaeren. Paris: C. Naud, Éditeur. 1903

[4] *L'Ordonnance du Tuberculeux.* Par le docteur Savignac. Paris: C. Naud. Price 30 centimes.

We have also received several small books on subjects connected with digestion and excretion. Dr. Aitchison Robertson has collected some valuable articles, already published elsewhere, dealing with the digestion and absorption of sugars,[3] and with the secretion of saliva. We commend the book to those who are interested in the physiological questions involved, as well as to all who have to treat defects in assimilation clinically. Some useful notes on a variety of infants' foods are contained in one of the papers.

Constipation[4] is always with us, and appears to be a special vice of civilised life. Dr. Sherman Bigg devotes a little treatise to a consideration of its causes and cure. A large number of formulæ for prescriptions are given at the end of the book. There is also advice as to massage and exercises in the relief of constipation, among which we may quote one consisting in squatting down and hopping round the room like a frog. This should at least add to the gaiety of domestic life. Dr. Leftwich regards the large intestine as a syphon,[5] an idea suggested perhaps by the shape of the viscus in the human being, but difficult to reconcile with the consistency of its contents. He also maintains, probably with reason, that during life the bands of longitudinal muscle in the wall of the colon are tonically contracted and prevent much alteration of shape. He gives some practical rules for treatment founded upon his theory, and insists on the danger of straining. In appendicitis the patient is to sit up in bed "as if he were orthopnæic (*sic*)." We do not feel convinced as to the correctness of either the theory or the practice.

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[1] *How to take Care of a Consumptive.* By M. Forrest Williams. London: John Long. 1903. Price 1s. nett.

[2] *Transactions of the American Climatological Association for the Year* 1902. Philadelphia: Printed for the Association.

[3] *Contributions to the Physiology of Digestion and to Dietetics.* By W. G. Aitchison Robertson, M.D., D.Sc., F.R.C.P.E. Edinburgh. 1903.

[4] *Constipation.* By G. Sherman Bigg, F.R.C.S. (Edin.), late Surgeon-Captain, Army Medical Staff, &c. London: Baillière, Tindall, and Cox. 1903. Price 2s. 6d. nett.

[5] *On Syphonage in the Large Intestine.* By Ralph Winnington Leftwich, M.D., late Assistant Physician to the East London Children's Hospital. London J. and A. Churchill. 1903. Price 3s. nett.

Muco-membranous colitis[1] is an obstinate disease, and one which is little understood. A short account of what is known about it is given by Dr. Froussard, of Plombières les Bains, one of the watering places which are recommended for the treatment of the condition. He regards the trouble as dependent upon a disturbance of the nerve-supply of the large intestine, a view which is supported by the occurrence of the malady chiefly in women and neurotic individuals.

Mr. Sim Wallace calls attention to the advantages to be derived from due performance of the process of mastication,[2] and to the drawbacks to the modern over-refinement of foods. He even has a good word to say for the domestic "comforter" which we have always regarded as an abomination. We must protest against regarding a piece of septic india-rubber, transferred from the infant's mouth to the floor and back again, with any quantity of dirt and germs adherent, as anything but a danger to health; but with Mr. Wallace's contention that children when they have got their teeth should be supplied with something to use them on—crusts and the like—we are in hearty sympathy.

To readers of a philosophical turn of mind who are conversant with the German language we would recommend a little book on Non-medicinal Remedies[3] by Dr. Buttersack. They will find there an interesting consideration of the ultimate causes of health and disease, and of the influence which we can hope to exert on vital activity by remedial measures.

[1] *Muco-membranous Colitis*. By Dr. Froussard. London: Baillière, Tindall, and Cox. 1903. Price 1s. nett.

[2] *The Physiology of Mastication and Kindred Studies*. By J. Sim Wallace, M.D., D.Sc., L.D.S., Hon. Dental Surgeon, West End Hospital for Nervous Diseases, &c. London: J. and A. Churchill. 1903. Price 1s. 6d.

[3] *Nichtarzneiliche Therapie innerer Krankheiten*. Von Dr. Buttersack. Berlin: Aug. Hirschwald. 1903. (Bibliothek von Kohler.)

The influence of mind on body, and the reäction of living organisms to various stimuli, are discussed in the first half of the book, along with a wealth of quotation from ancient and modern philosophical authorities which is fascinating in itself. The second half of the volume is devoted to a brief account of such methods of treatment as light, X-rays, gymnastics, &c. An interesting retrospect of the progress of medicine and of its dependence upon contemporary scientific speculation is to be found in a "Festrede" by Professor Leyden,[1] recently published. The writer enforces the point of view, sometimes apparently lost sight of in these days of research, that after all the object of medicine is the cure of sick human beings; it is an art, not a pure science, as Hippocrates long ago recognised.

Medical students undoubtedly need assistance in their studies, but we doubt if they are much the better for such aid as is supplied in the shape of cram-books. Messrs. Livingstone, however, think otherwise, and are issuing a series of small volumes for the use of those about to undergo "the question." Their "Catechism Series"[2] now contains sets of questions on Physiology, Pathology, and Physics. The information given seems to be reliable. That such books are needed or sought for is the fault of the modern system of examination, which encourages a memory for facts rather than a knowledge of principles and their application.

Three Dictionaries of Medicine have lately come to hand. Of these the largest and most important, compiled by Dr. Dorland, is an illustrated volume[3] of nearly 800 pages, constituting a valuable work of reference. It appears to be very comprehensive and complete, and contains useful tables of muscles, nerves, &c., as well as definitions of medical terms drawn from far and near. There is a very complete list, for instance, of the different diseases which are called after the names of their discoverers, of which there are now so many

[1] *Das Denken in der heutigen Medicin.* Festrede von Dr. E. von. Leyden. Berlin: Hirschwald. 1903.

[2] *Catechism Series.* Edinburgh: E. and S. Livingstone. (Physiology, Parts I.-IV.; Pathology, Parts I.-III.; Physics, Part I.) Price 1s. nett each part.

[3] *The Illustrated Medical Dictionary.* By W. A. Newman Dorland, A.M., M.D. 3rd edition. 8vo. Bound in flexible morocco. W. B. Saunders and Co. 1903 Price 19s. nett.

that it is difficult, if not impossible, to remember the majority of them. The illustrations are also good, though we doubt whether pictures showing the histology of tuberculosis should be included, unless other morbid conditions receive equal treatment, which would be practically impossible. A smaller dictionary[1] by the same author is also issued, which likewise seems accurate and full. A dictionary on different lines[2] is issued by the same firm of publishers, the apparent object of which is to supply the reader with the complicated and generally barbarous synonyms used presumably to conceal our meaning in speaking of medical matters. We are not sure that any useful purpose is served by such a work. For example, we cannot quite see what anyone who is writing or speaking of the existence of hydrogen sulphid(e) in the blood—if it exists there—would gain by learning that it had been called "hydrothyonemia," that a rust-coloured substance may be called "fusoferruginous," that a wound which heals with difficulty is "dysapulotus," or that an agent which stimulates the muscle of the heart is an "angiocardiokinetic." The only useful feature which we have discovered in the book is a list of common drugs with their scientific names. Another small work of the "Dictionary" order is Dr. Stretch Dowse's *Pocket Therapist*,[3] which has reached a third edition. It contains a great deal of information, of somewhat unequal merit, on medical matters, and may be useful occasionally to the busy practitioner in doubt as to treatment. We doubt the advisability of a classification which puts "Perforation of the intestine" under "Hæmatemesis," and omits any cross-reference under the former heading.

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[1] *The Pocket Medical Dictionary.* 4th edition. Pp. 556. 5s. nett.

[2] *A Thesaurus of Medical Words and Phrases.* By Wilfred M. Barton, M.D., Assistant Professor of Therapeutics, &c., in Georgetown University, and Walter A. Wells, M.D., Demonstrator of Laryngology, Georgetown University, &c. Pp. 534. W. B. Saunders and Co. 1903

[3] *The Pocket Therapist: A Dictionary of Disease and its Treatment.* By Thomas Stretch Dowse, M.D. (Aberd.), F.R.C.P. (Edin.). Bristol: John Wright & Co London: Simpkin, Marshall, Hamilton, Kent & Co., Ltd. 1903. Price 6s. 6d nett

Notes by the Way.

Medical Education.

THE question of education, medical and other, has been so much in evidence—it might seem impolite, though hardly inappropriate, to say that it has been and remains "in the air"—that no apology is necessary for recurring to the subject. It is indeed one of paramount importance to the profession and the public, for the efficiency of the practitioners of the future depends largely on the training which they are compelled to undergo in their youth. Hence it is an urgent duty to ascertain whether the training now afforded is the best that can be devised. Having ourselves entered rather late upon the peculiar obstacle-race which is called the medical curriculum, after wandering for a fairly lengthy course in the gentler paths of classics and philosophy, we feel that we may speak with some degree of impartiality of methods of education. Certainly, what strikes us with regard to the present system is the cast-iron rigidity insisted upon in the matter of the training of every budding Hippocrates. Every candidate, whatever his place of study and previous up-bringing, must go through the same identical course of preliminary sciences when he enters as a student, before he begins the studies really essential for the pursuit of medicine. From this it must be concluded that the reigning authorities consider that the smattering of many sciences insisted upon is of indispensable importance to the future doctor. This we emphatically decline to believe. We do not imagine that anyone will be the more competent to treat a sick man because he has learnt to look for sieve-tubes in a plant or to describe the spiral valve in the dog-fish. We are strongly of opinion that the vast majority of medical students forget most of the preliminary science which they have learnt, long before they reach the final examination. In other words, the actual facts laboriously acquired at this period of study are of no ultimate value.

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Mental Gymnastics.

BUT it may be said that these preliminary studies are not intended to store the memory with necessary facts, but to afford a general education—to train the mind to assimilate other matter later. In that case it is evident that, as these sciences do not constitute the only means of supplying the required mental gymnastics, there ought to be some means by which those who have had their assimilative faculties cultivated by other means may be enabled to proceed to the really necessary studies without delay. The preliminary examinations in some at least of the non-medical sciences, if we may so call them, should be excused in those who have passed a sufficiently stringent test of general knowledge to prove that they are so far educated as to be capable of appreciating the truths of the indispensable sciences. If such a regulation should result in greater attention being paid by students to such subjects as writing, spelling, and composition, with a corresponding diminution in their acquaintance with botany or animal morphology, we believe that we should have attained "a consummation devoutly to be wished."

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The Irreducible Minimum.

WHAT, then, are the actual sciences which are indispensable for a medical man? In the first place, Chemistry is absolutely necessary, if we are to escape the charge of "pouring drugs of which we know little into bodies of which we know less." It must include the study both of inorganic and organic matter, and of physiological processes, so far as they are known; but we believe that the former branch might be made much more practical than is done at present, and include what is necessary of pharmacology; and that much more stress might be laid on the study of the vital processes which are of so great an importance in actual practice. A second indispensable subject of study is Anatomy, which, again, should be taught practically, not pedantically—and on this point we may call attention to the weighty criticisms of modern methods from the pen of "An Old Teacher," which appear on another page. Thirdly, Physiology is the basis of all medical knowledge, and to it the greatest possible attention should be given. Yet even here there is scope for a wise selection of the subjects to be mainly insisted upon. We feel sure that time spent in mastering the details of complicated apparatus, adapted to measure and record vital phenomena, is time wasted in the case of the average student. All the minutiæ, too, of the experiments which have been made on "muscle and nerve," however fascinating to their authors and to "Honours men," are beyond the limits which separate the needful from the ornamental part of physiological knowledge for a medical man. If such matters were omitted—at least as subjects of examination and its concomitant cramming—more attention could be paid to the great functions with which medicine is chiefly concerned, digestion, secretion, reproduction, and—when physiologists at last come to recognise it—the influence of the nervous system on all vital functions and all cellular activity in man.

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Science at School.

FROM what we have said with regard to the preliminary sciences, it naturally follows that, so long as they are maintained as integral parts of medical education, we sympathise with those who hold that they should be got through at as early a stage of the student's career as possible. They are not indispensable, and the sooner they are in any manner cleared out of the way the better. Hence a candidate should be permitted to present a certificate of having attained the requisite degree of knowledge in these subjects at school, and should then be excused from further study of botany or zoölogy; and even elementary chemistry can be as well learned at school as at a medical college. It is difficult to see why any wish should exist to prevent our students from clearing their preliminary obstacles at as early a stage as possible. It must, however, be clearly understood that such scientific work at school must not be allowed to interfere with the attainment there of what may be called general culture, meaning, above all things, writing, spelling, and English composition. "These things ought ye to have done, and not to have left the others undone." Who can doubt that the curriculum, as at present constituted, is too long and too heavily loaded? The attention of our task-masters should be turned to the question of lightening it of all unnecessary matter. With this object, in view of the ever-growing demands of the actual art of medicine upon the time of its votaries, all preliminary studies should be cut down to the lowest possible quantity, and left behind at the earliest possible moment.

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A Paper Scheme. As there is no probability that anything that we may say will very immediately influence the course of events, which lies upon the knees of the General Medical Council, we can fearlessly propound theoretical solutions for our present evil plight. Theory then would seem to suggest that a medical student should at the outset of his career have to pass a matriculation examination in matters of general culture, including elementary chemistry, and perhaps elementary physics, before being registered. This examination should constitute a test of the possession of a degree of polish adequate to the needs of a member of a learned profession. He should then devote the first part of his time to the study of more advanced chemistry, of anatomy, and of physiology, all of which should be taught practically for his needs, not in any degree as mere education or mental gymnastics, still less with a sort of rivalry between the different teachers, each claiming for his own subject the greatest possible amount of the pupil's time. Then after passing an examination in these three subjects, which might be conducted by the separate teaching institutions under central guidance, the student should proceed to the study of the art of healing in the wards of a general hospital. This second stage, again, would be terminated by an examination, which would qualify for practice. This final test might with advantage be conducted by a single central authority, which would hold examinations in the different medical centres, thus affording a uniform standard for all the country—the "one portal" which has often been desired. University degrees should not qualify for practice apart from this examination, but should constitute additional qualifications guaranteeing the possession of special knowledge as tested by a higher standard, analogous with the "F.R.C.S." and "M.R.C.P." as they now exist. A *reductio ad absurdum* of the attainment of a qualification by a university degree alone is afforded by the University of London, which does not exact a knowledge of

Latin in its matriculation. Hence an M.D. of this university might never have learnt the rudiments of a language in which he will write prescriptions, and in terms of which many parts of the body are named by anatomists.

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The Use of Lectures.

WE cannot help thinking that under the existing arrangements too much stress is laid on the attendance of students at lectures and too little on their work in the wards of a hospital or in the out-patient department. It may well be doubted whether, in the majority of subjects, lectures are of much practical use, now that so many excellent text-books are available. The theory of medicine does not lend itself more readily to oral exposition than to reading, as far as we can see; and much of the time spent on listening to lectures might with greater profit be employed in gaining a practical knowledge of the treatment of living patients with which the work of a doctor's life is concerned. This might not lend itself so kindly to the system of "signing-up," so dear to the hearts of the powers that be, but it might produce results better for the patients destined to fall into the hands of newly-qualified practitioners. We have nothing to say against the insistance on a certain number of certificates showing that the candidate has attended courses of special instruction in such subjects as fevers or insanity; but the mere attendance at lectures proves nothing which should not be capable of being tested by the examination itself, to which the "signing-up" is a preliminary ceremony.

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Machine-made Diagnosis.

WHETHER it be due to our methods of teaching or not, we cannot say, but there can be no doubt that the tendency of the present time is to rely more and more upon laboratory methods of diagnosis and less upon clinical examination of patients. If we are in doubt as to whether a patient is suffering from diphtheria, we care little as to the presence or absence of membrane in the throat; we send a "swab" to the nearest laboratory, and make our diagnosis on the report of a man who has never seen the case at all. If enteric fever is suspected, the blood must be examined; and as the Widal test is positive or negative, so the sufferer's ailment is classified. It is very convenient, no doubt. The onus of a decision is thrown on to some one who has no responsibility in the matter, and we are free from blame in any case. And so great is our faith in the refinements of modern laboratory methods that their findings are often accepted in the face of distinct clinical evidence to the contrary. Yet we would venture to suggest that no single one of the laboratory tests has as yet established itself as infallible. It is known that virulent diphtheria-bacilli may exist in healthy throats, and therefore also in sore throats which are not diphtheritic. Agglutination of *B. typhosus* may occur with serum from patients who are not suffering from enteric fever, and may fail to appear in the case of those who are so afflicted. In short, the tests are useful in their way, if used rationally as aids, of quite subordinate value to the study of clinical symptoms and signs. Otherwise employed, to the neglect of the phenomena met with in individual cases of illness, they are as likely to mislead as to help the too confiding practitioner.

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Death-certification.

We alluded last year[1] to the recently-issued list of causes of death as classified by the experts of the Registrar-General's office. We have now received a very instructive and interesting Blue Book[2] dealing with the statistics for the year 1901, and containing a letter addressed by Dr. John Tatham, F.R.C.P., to the Registrar-General. A brief account is here given of the history of death-registration, for the setting of which upon a scientific basis the country is practically indebted to the late Dr. William Farr. His work has been worthily carried on by his successor, Dr. Ogle, and lately by our distinguished colleague, Dr. Tatham. The tabulation of causes of death is not so simple a matter as might at first sight appear. It is true that the College of Physicians prepares an authoritative list of the names of such diseases as in their opinion we are to be allowed to suffer from;

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[1] THE PRACTITIONER, August, 1902, p. 138.

[2] *Sixty-fourth Annual Report of the Registrar-General of Births, Deaths, and Marriages in England and Wales* (1901). London. 1903.

but not all of these are fatal, so that a selection has to be made of such as are likely to result in death, and these, for convenience of statistical presentation, have to be divided into groups. These groups continually alter with the extension of our knowledge of pathology. Thus it is necessary to determine whether to class "acute pneumonia" as a local or a general disease; whether to put tuberculosis among infective processes; whether apoplexy is a disease of the nervous system or of the blood-vessels. The answers to these questions may and do vary from time to time, and hence the classification adopted in the Registrar-General's office also varies. Names also alter. Thus, epidemic Rose Rash now appears as German Measles, though we do not know whether this change points to improved relations with our Continental rivals or the reverse; Remittent Fever is now called Malaria; and Sarcoma is included under Malignant Disease, as distinct from Cancer. Much interesting material with regard to the prevalence, or at least the fatality, of different diseases is to be found in this Blue Book, which we commend to the notice of our readers.

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Inebriety in Women.

THOSE who are interested in the social effects of alcoholic indulgence will find some important papers on the subject in the October number of the *British Journal of Inebriety*, which contains a series of articles on the disease as it occurs in women. We say "disease" advisedly, as we are convinced that it is time that the State recognised that the unfortunate beings who become slaves to alcoholism are no longer able in most cases to resist the craving, and are no more to be treated as criminals than others who suffer from well-known mental defects. The light and shade of the subject are differently represented in the different articles. The most hopeful, perhaps, is one written by Mr. Waugh, of the Society for the Prevention of Cruelty to Children, who explains the measures taken by his society to encourage the drunken mother to reform, by means of sympathy and encouragement. The gloomiest view of the female inebriate is afforded in a paper by Mr. Holmes, the well-known police-court missionary, who shows that of the many women who figure on the "black list" a very large number are quite incapable of reform—mentally-defective creatures in whom the indulgence in strong drink is part of a sensual nature, leading inevitably to prostitution and moral degradation. "I know that bolts and bars will not cure them," writes Mr. Holmes, "that two years, though spent in retirement and healthy surroundings will not reform them; that human sympathy, coupled with earnest effort and prolonged care, will not make them as other women. To God and science their future belongs, for they cannot help themselves." It is this inability to help themselves that constitutes the difficulty in reforming the inebriates of either sex. They have not the will-power to resist the alcoholic craving. The great defect in the present state of the law as affecting confirmed drunkards is the lack of power to place them, even against their will, in some reformatory home, if possible before the evil has become incurable. Until such compulsory powers exist in the case of inebriates as of other mentally-defective persons, we fear that little good will result in the direction of protecting either the drunkards from themselves or their unfortunate families from the ruin which their insane conduct causes.

EVERYDAY AILMENTS AND THEIR TREATMENT

BRONCHITIS.

IN considering the treatment applicable to a case of bronchitis it is necessary to bear in mind the ætiology of the disease. In a very small number of instances it may be caused by irritating vapours or solid particles acting directly on the mucous membrane of the bronchial tubes. Perhaps a cold east wind may thus directly excite catarrh in mouth-breathers. In ordinary cases the disease is due to the action of microörganisms, which possibly exist as harmless saprophytes in the air-passages under conditions of health, and are enabled to become pathogenic by some diminution of the normal resistance of the tissues. It is thus in all probability that a chill is effective in exciting bronchitis. Chronic Bright's disease and lesions of the cardiac valves act as predisposing causes, owing to the defective nutrition of the bronchial mucosa brought about in the one case by poisons in the blood, and in the other by constant passive hyperæmia. In other instances the disease is produced as a symptom or complication of an infective disease, such as measles or influenza, either by the direct action of the causal organism or its toxines, or, as above, by facilitating secondary infection, when the resistance of the bronchi is lowered by the infective process.

Pathologically, the first stage of an acute attack of bronchitis is marked by dryness and swelling of the mucous membrane. The lining of the tubes is hyperæmic and secretes only a small quantity of tough mucus, which may be streaked with blood. A little later the secretion becomes more free, the cells of the epithelium undergoing mucoid degeneration, while leucocytes tend to escape on to the surface in increasing quantities, giving the expectoration its mucopurulent and even purulent character. In acute cases which recover, the phenomena of inflammation subside as they appeared, and the mucosa regains its normal state. If, however, as the result of repeated attacks or from some defect in resistance the condition becomes chronic, there results a thickening of the submucous tissues, while the overlying epithelium is largely destroyed. Owing to this damage and to the persisting inflammation, a continuance of the secretion is seen, the sputum being copious and mucopurulent. Complete recovery seldom or never occurs when the disease has once reached this stage.

A certain degree of pulmonary emphysema is generally present in chronic cases, being produced by the constant cough, acting upon alveoli which are in many cases deficient in elastic substance as the result of degenerative processes accompanying chronic renal disease or cardiac lesions.

We thus see that the indications for treatment vary considerably in the different stages of the disease. In the early phase, the patient complains of pain or soreness in the chest; the sputum is scanty and brought up with difficulty, while there is often dyspnœa from narrowing of the tubes by swelling of the mucosa and adherent plugs of mucus. The physical signs produced are rhonchi of varying pitch, formed as the air passes through constricted portions of the air-ways. Under these circumstances much may be done by supplying the inflamed tubes with warm, moist air, which has a soothing effect and counteracts the distressing sense of dryness in the chest. A steam-kettle and a tent-bed often give great relief. Warm fluid food should be given, as swallowing hot drinks has a reflex effect on the bronchi, and aids the expectoration, apparently by increasing the flow of mucus. Dr. Mitchell Bruce advises the use of hot lemonade (home-made) drunk freely. Hot poultices or fomentations to the chest and back are comforting to most patients; in other cases stimulating liniments, such as that containing turpentine and acetic acid, may be preferred. By way of medicine we may give either such a mixture as: Ammonium carbonate, 3 grains; iodide of potassium, 2 or 3 grains; solution of acetate of ammonium, 2 drachms; and camphor-water to 1 ounce: to be taken every four hours. Or: Ipecacuanha wine and tincture of squill, of each 10 minims; spirit of nitrous ether, ½ drachm, and chloroform-water to 1 ounce. The effect of iodide of potassium in small doses is to produce a thinning of the bronchial mucus, and it is thus sometimes effective in cases in which the sputum is tough and difficult to bring up. It is well to remember that if it be desired to add this drug to the second of the mixtures given above, it is necessary to add at the same time a little alkali of some sort, such as ammonium carbonate, to neutralise the slight acidity of the nitrous ether.

In old persons there is often a tendency to heart-failure accompanying bronchitis. This may call for the exhibition of stimulants, such as alcohol or strychnine, the latter being administered preferably hypodermically. Or a mixture containing ether and ammonia may be employed, *e.g.*, spirit of ether (sulphuric, not nitrous) 20 minims ; sal volatile, ½ drachm ; syrup of orange, one drachm ; and camphor water to one ounce. This may be given every four hours, and if there is need, a few drops of tincture of digitalis may be added to it.

If the bronchitis continue, and there are cyanosis and signs of engorgement of the right side of the heart, venesection may be needed and often gives great relief, eight or ten ounces of blood being taken from the median basilic or any other convenient vein. Instead of this, if there is any reason to hesitate to bleed, leeches may be applied over the front of the chest, and the hæmorrhage encouraged by hot fomentations.

A brisk purgative is often useful at the outset of a case of acute bronchitis. Carlsbad salts or a Seidlitz powder may be used, with or without a preliminary dose of calomel (2½ or 3 grains). The use of sedatives, such as opium or morphia, is not to be recommended in bronchitis, since not only do they reduce the sensitiveness of the bronchial mucous membrane and thus prevent the irritation which is the basis of cough and so of the necessary expulsion of mucus, but the secretion from these surfaces is diminished in amount and rendered more viscid, so that it is brought up with greater difficulty, while the respiratory centre in the medulla is depressed, and any tendency to asphyxia aggravated. Hence any sedative drugs must be given with caution and only in suitable cases. Any lividity or sign of respiratory failure is an absolute contraindication. Probably the only condition in which an opiate linctus is permissible is when there is an irritable, dry cough, which is not effectual in bringing up any sputum and which prevents sleep. If the patient is robust, the heart working well, and respiration not embarrassed, a few drops of the liquor morphinæ may be permissible, or we may employ instead the morphine-derivative, heroïn, in doses of $\frac{1}{16}$th to $\frac{1}{10}$th grain. As a general rule we may, however, recall the ancient tip that morphia is to be avoided in the "Three B's"—Bright's disease, bronchitis, and babies.

In young children the course of treatment may be on the same lines as have been sketched above, but it is generally better not to employ poultices to the front of the chest owing to the softness of the ribs, and the necessity of avoiding any impediment to the respiration in the form either of weight or constriction. A light jacket of cotton-wool may be used instead, the chest being well rubbed back and front with a liniment of turpentine and acetic acid of half the strength of the official preparation. An emetic is sometimes useful in clearing away the mucus from the air-passages, if there is much cyanosis and labour in breathing; but it is not usually called for, and it must be remembered that a depressant like ipecacuanha, the usual emetic, is to be avoided if possible.

The danger in bronchitis, unlike that in croupous pneumonia, is rather the mechanical blockage of the bronchial tubes with mucus, whereby the entry of air is impeded, than a toxæmia due to infective organisms. The patients often tend to lie propped up in one position, without change of posture, and may in bad cases sink into a drowsy condition, so that the mucus tends to accumulate in the lower air-passages and to leave portions of the lung imperfectly expanded and aërated. For this reason, as has been pointed out by Bruce, the process of making a patient sit up in bed for percussion and auscultation is often beneficial, and we need not hesitate to make a daily physical examination of the bases of the lungs, although it may cause coughing. This is really an advantage, and the patient's ultimate good should be thought of, rather than his immediate comfort.

As the case progresses, the sputum becomes looser, and the poultices to the chest may soon be left off. If a stimulating liniment have been used, it may be presevered with during the second stage of the disease; but it is well not to pass directly from poultices or fomentations to a liniment, as the skin may be unduly tender at first after the moist applications. The steam kettle may next be dispensed with, and then the tent gradually removed, the curtains being first looped up, if the weather be cold, before the whole is dispensed with.

By continued use of an expectorant or alkaline mixture exudation into the bronchi can be kept up indefinitely. It is necessary, therefore, during convalescence to discontinue the use of such medicines, and the disappearance of moist sounds from the chest may be accelerated by administering an acid tonic, such as dilute nitro-hydrochloric acid, 10 minims; tincture of nux vomica, 5 minims; spirit of chloroform, 10 minims; and compound infusion of gentian to 1 ounce; to be taken three times a day after meals. Or: tincture of cinchona, 20 minims; dilute nitric acid, 10 minims; syrup of orange, 1 drachm; and chloroform-water to 1 ounce, may be substituted. In some cases quinine may be useful, in others some form of iron-preparation is indicated. In children a course of the compound syrup of the phosphate of iron is often useful, or malt-extract and cod-liver oil may be employed.

CHRONIC BRONCHITIS.

When bronchitis has become chronic, the condition of the air-passages is no longer susceptible of complete *restitutio ad integrum*. A certain amount of damage to the mucous membrane will remain in any case, and act as a predisposing cause of subsequent attacks. Some secretion exudes almost continually, and on the slightest provocation a subacute bronchitis is lighted up. The expectoration is usually muco-purulent, and often reaches a large amount. Some degree of emphysema is almost invariably present in addition. Unless a more than usually acute exacerbation occur, there is little need of expectorants; but should such an acute attack occur, it must be treated as sketched above. Otherwise most may be done for these patients by attending to the general health. If circumstances permit, it is desirable for these sufferers to winter in a warm climate, so as to avoid the cold and damp of this country. Needless to say they should not return too soon in the spring, which is often more inclement than the nominal winter. The appetite must be encouraged, and cod-liver oil and preparations of malt are as suitable for these patients in many cases as for the tubercular.

No special limitations as to diet are necessary in these cases, as a rule, any good nourishing form of food being permissible. It is well to remember, however, that in a certain proportion of cases of bronchitis there is a gouty factor, and that in such instances an "anti-gouty" regimen must be adopted, a mixture containing bicarbonate of potassium and a little iodide of potassium, with sulphate of magnesia to regulate the bowels, sometimes acting very beneficially on the bronchial condition.

Owing to the frequent coëxistence of chronic renal disease with chronic bronchitis, it is well to make a point of examining the urine for albumen and casts in all these cases.

Novelties and Notices.

"TABLOID" SPARTEINE SULPHATE.

THE action of sparteine is comparable to that of digitalis, the drug being useful in some cases of heart-failure when digitalis is unsuccessful. Messrs. Burroughs, Wellcome & Co. have for some time manufactured half-grain tabloids of sparteine sulphate for hypodermic use, and they have now introduced a one-grain tabloid for administration by the mouth. Sparteine has not, perhaps, yet found its way into very general favour, but it is deserving of trial in cases in which no good results ensue on the administration of the more common cardiac tonics.

HEMISINE.

The name "Hemisine" is given to a solid preparation of extract of suprarenal gland, which is manufactured by Messrs. Burroughs, Wellcome & Co. for use as a hæmostatic. "Soloids" containing 0·0005 grm. weight are prepared, suitable for dissolving in 5 cc. (84 minims) of water, the resulting solution being of a strength of 1 : 1000. The use of suprarenal extract in nasal and ophthalmic work is well established, and the convenience of having a solid preparation instead of the ordinary fluid extracts, which keep badly, is obvious.

FRY'S COCOA AND MILK.

This preparation of cocoa with condensed milk and sugar contains in a small bulk a large amount of nourishment, and is very convenient for transport. A teaspoonful of the cocoa and milk added to a cupful of hot water makes a most agreeable beverage of excellent flavour, without the need of any additional milk or sugar. To those who like their cocoa sweet we can safely recommend it. In case of the absence of hot water the preparation can be eaten alone, and forms a not-unpalatable sweetmeat, as well as a nourishing food.

PLASMON ARROWROOT.

Plasmon has now established itself as a valuable food-stuff, and is obtainable in various forms—biscuits, cocoa, &c. The latest preparation which we have received is Plasmon Arrowroot. The addition of plasmon—an albuminous substance, obtained from milk—to the starchy material much increases its nutritive value. For use, three teaspoonfuls of the arrowroot are mixed with half a pint of warm milk or water, and boiled for two minutes. It should form a useful invalid food, if used in suitable cases. It is also claimed that plasmon arrowroot is of use in certain forms of diarrhœa. It may be recommended as a valuable food for convalescents, during the period of transition from "slop diet" to normal solid food.

Practical Notes.

CAUSES OF FAILURE OF DIGITALIS.—In a lecture published in the *Presse Médicale* (July 4, 1903), Dr. Barié gives some of the reasons which may account, in particular instances of cardiac disease, for a failure to reäct to digitalis. The special indications for the drug are well known to be frequency and irregularity of the heart-beat, with diminution of arterial pressure; while serious degeneration of the myocardium is generally a contra-indication to its employment. In cases of cardiac failure apparently suitable for treatment by digitalis, gastric derangement may prevent our making use of it, as it may cause vomiting. In such patients a preliminary purgative dose of jalap or sulphate of magnesia may pave the way for the use of digitalis. Barié recommends the compound tincture of jalap of the German Pharmacopœia for employment in such cases. In some patients it is necessary to persist in a pure milk-diet all the time that digitalis is administered, milk by its powerful diuretic action and owing to its lack of sodium chloride constituting a valuable auxiliary to the drug. As much as three litres (about five pints) of milk must to be taken to constitute sufficient food for an adult. In other cases accompanied by great œdema, digitalis cannot act until the venous circulation has been relieved by a preliminary venesection, which should amount to about ten ounces of blood; or Southey's tubes may be used to drain off the accumulated fluid; or multiple punctures be made through the skin. Peritoneal or pleural exudates should be similarly drawn off, if they have reached a considerable quantity. In all or most cardiac cases there comes a time when digitalis fails to act, owing to myocardial degeneration, its action seeming to get feebler and feebler in successive attacks of failure of compensation. With regard to administration, Barié recommends the use of digitalis for periods of five or six days, with intermissions between, in order to avoid any cumulative action. He also endorses Potain's rule as to its employment, viz., to continue the digitalis as long as there are traces of œdema remaining anywhere. A useful method of administering the drug in many cases is that of Pfaff, which consists in giving large doses at first, and then gradually diminishing them on succeeding days. This plan, however, should not be adopted when there is tricuspid insufficiency, which is acting as a safety-valve. Small doses are also to be used in cases in which compensation is well established, and the drug is only given as a tonic for the relief of functional irregularity. Special care is necessary in prescribing digitalis in the case of old people with degenerate arteries. In aortic regurgitation digitalis is contra-indicated, as tending to prolong diastole, thus permitting greater regurgitation, and also as tending to constrict the peripheral arterioles and so to increase the work thrown on the left ventricle. The peculiar condition of the heart-beat manifested by the so-called *pulsus bigeminus* is a signal to refrain from the use of digitalis, as in some of these cases sudden death has occurred after its administration. The appearance of this symptom during treatment calls for a discontinuance of the remedy. Barié enumerates as drugs which are antagonistic to digitalis and which, therefore, should not be employed along with it: opium, alcohol, alkaline iodides, most vaso-dilatators, diffusible stimulants, and astringents. In the case of some at least of these remedies we do not think that the antagonism is absolute; thus in many cases alcohol is beneficial if given between the doses of digitalis, and iodide of potassium seems to fulfil a distinct indication in cases of aortic disease associated with cardiac failure, in that it dilates the arterioles, thus counterbalancing one of the ill effects of digitalis as applied to this condition. Nitro-glycerine, another vaso-dilatator, may be useful in these cases for the same purpose, the digitalis meanwhile stimulating the cardiac muscle.

SANTONIN IN DIABETES.—"Of all the medicaments which have been tried in diabetes none has succeeded better than santonin. It was studied by Dr. Sejournet, and systematically employed by him as a remedy for diabetes mellitus, with constant and indubitable benefit. Santonin, however, must not be given as a routine remedy; its use should be continued only till a sensible improvement is effected, which usually occurs within about a fortnight." (*Le Mois Thérapeutique.*)

SYPHILITIC ALOPECIA. — Besnil (*Journal Médical de Bruxelles*) advises cutting the hair short and the use twice a week of the following :—

℞ Pure oil of cade - 3 grammes (45 grains);
Precipitated sulphur - 1 gramme (15 grains);
Resorcin - 30 centigrammes (4½ grains);
Salicylic acid - 15 centigrammes (2¼ grains);
Benzoinated lard - 30 grammes (1 ounce).
M. For an ointment.

The morning after using the above, the scalp should be washed with hot water and ichthyol soap. (*N. Y. Med. Journ. and Philad. Med. Journ.*)

ARISTOCHIN IN WHOOPING-COUGH.—Kittel (*Therapeutische Monatshefte,* 1903, p. 408), states that he has found great benefit from the use of this quinine-derivative in whooping-cough, as recommended by Stursberg. The dose is regulated by the following formula :—As many centigrammes are to be given three times a day as the child is months old, if an infant; for older children 0·2 gramme may be given for a dose. The drug may be administered in water or milk, and is readily taken by children. It does not seem to give rise to any ill-effects. Out of thirty-four cases thus treated, twenty-five were cured, and seven others improved. The duration of the disease is shortened by the remedy, which appears to act most beneficially in infants under one year old. Appetite increases under its use, and the general condition is improved. Kittel considers aristochin a valuable addition to our means of treating whooping-cough.

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CORRIGENDUM.

On page 743 of our November number, line 15, for "palmation" *read* "palmitin."

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